A2623-1

merican Journal
of Public Health

May 1991 Supplement Volume 81 Established 1911

New York State HIV Seroprevalence Project

SUPPLEMENT

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American Journal of Public Health

Supplement

Compiled by New York State Department of Health

Lloyd F. Novick, ***Editor***

American Public Health Association

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©AMERICAN JOURNAL OF PUBLIC HEALTH May 1991, Volume 81

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The Journal, established in 1911, publishes reports of original research, demonstrations, evaluations, and other articles covering current aspects of public health. Neither the Association nor the Editors assume responsibility for the opinions expressed by authors of papers published or quoted in this Journal. Copyright 1991 by the American Public Health Association, Inc. Second class postage paid at Washington, DC and additional mailing offices.

Subscription price of the *American Journal of Public Health* is $80 per year domestic; $120 per year foreign (air mail); $10 prepaid for single copies of an issue or supplement from the American Public Health Association, 1015 Fifteenth Street, NW, Washington, DC 20005.

I. New York State HIV Seroprevalence Project: Goals, Windows, and Policy Consideration

Lloyd F. Novick, MD, MPH

Introduction

In the summer of 1987, the New York State Department of Health embarked on an extensive effort to determine the prevalence, distribution, and trends of human immunodeficiency virus (HIV) infection in the population. Statewide serosurveillance for antibody to HIV would be conducted by blind testing of available blood samples. Six populations were selected as "windows" on the epidemic: newborns, homeless adolescents, prisoners, and clients of family planning, sexually transmitted disease (STD), and drug treatment clinics. For each of these windows the objectives were to: 1) establish the baseline prevalence and geographic distribution of infection; 2) monitor trends of infection overall and in various populations, including age and racial/ethnic groups; and 3) use the information to target and design preventive activities.

Testing of all newborns in New York State began in November 1987, and all six study windows were operational by June 1988. As of March 1990 blind testing for HIV antibody had been performed on more than 725,000 blood specimens in these six studies. Two years of testing have yielded sufficient data for reliable analysis and projection of trends. The following chapters describe the methods and results of these seroprevalence activities, with emphasis on HIV infection in women of reproductive age, adolescents, and children. HIV seroprevalence rates are correlated with other socioeconomic indicators, including drug-related hospital discharges, the educational level of childbearing women, and the incidence of low birthweights. Information from these surveys has been used to identify high HIV infection rates in small geographic areas and to spur preventive activities statewide.

Impetus for Statewide HIV Serosurveillance

These serosurveillance activities were prompted by two phenomena: the large number of individuals with clinically apparent AIDS in New York State, and the likelihood of a much larger number asymptomatically infected with the virus.

Since the first cases of AIDS were described, New York State has reported more cases than any other state[1] (29,186 by March 31, 1990).[2] Although 84 percent of these cases are residents of New York City, a substantial number (3,564) reside elsewhere in the state. By the same date a total of 18,239 deaths were attributed to AIDS.[2] In New York City, AIDS is the leading cause of death for males 30 to 49 years old, the leading cause of death for females between 20 to 39 years old, and the second leading cause of death for males 20 to 29 years old.[3]

Among children 648 cases of AIDS were reported in New York State as of March 31, 1990.[2] These case reports represent only a fraction of HIV-infected children in New York State, as estimated in the Newborn HIV Seroprevalence Study (see chapter II).

Although case-report data were invaluable in plotting the rise in incidence of AIDS and delineating the associated risk factors, severe deficits in understanding of the epidemic remained. Above all, the current distribution of HIV infection could not be determined from case reports. Most individuals infected with HIV are asymptomatic and not likely to be reported to public health departments, even in states which mandate reporting of HIV infections. (New York State requires reporting of AIDS cases but not HIV infections.) AIDS case reports also lag far behind the prevalence of HIV infection. The median lag time between infection and development of AIDS, resulting in a report to a public health agency, may be 10 years or more.[4]

To determine the actual occurrence of infection, direct and contemporary measures of those infected were needed. The multi-window HIV serosurveillance program could provide such a measure.

A critical need in planning public health interventions was to determine the extent of HIV infection in the entire community and in definable groups and locations. A comprehensive monitoring system, based on a representative, continuing serosurvey, was also needed to track the trends statewide and for age, racial/ethnic, and other groups on a statewide, regional, and community basis.

Approach to Statewide HIV Serosurveillance

The seroprevalence survey method adopted by the New York State Department of Health was blind testing of available blood specimens for antibody to HIV. Representative sampling of households was rejected, although the National Center for Health Statistics and the Centers for Disease Control used this method for a national survey designed to reach 50,000 respondents.[5,6] Seroprevalence analysis of available blood specimens could be implemented faster and more economically than a household survey and would avoid the problems of self-selection and bias associated with community surveys, which require voluntary participation and consent.

Before the Department of Health's blind seroprevalence studies were initiated, extremely limited data were available on the extent of HIV infection in New York State. Seroprevalence information was limited to military recruits and blood donors. Of recruits from New York State 0.35 percent were seropositive, 0.36 percent of males and 0.28 percent of females (Centers for Disease Control: Quarterly Data Report on HIV in Civilian Applicants for Military Service, June 22, 1989). Of 134,144 first-time blood donors seen between July 1987 and September 1988, 0.09 percent were seropositive, 0.12 percent of males and 0.06 percent of females.

The New York State program was based on six major windows, i.e., population groups from whom blood specimens are routinely collected for other purposes. In virtually all cases, sufficient specimen remains for blind assay of antibody to HIV. The six windows are (Table I-1):

Newborns: A dried-blood specimen is routinely collected from every infant born in New York State and screened by the State Department of Health for seven inherited metabolic disorders (see chapter II).

Family planning clients: A number of family planning clinics routinely collect blood specimens for syphilis testing as part of the client's initial medical examination (see chapter VII).

Runaway and homeless adolescents: A shelter for high-risk adolescents in New York City routinely collects blood specimens from individuals referred for medical examination (see chapter IX).

STD clinic clients: Blood is routinely drawn at sexually transmitted disease (STD) clinics for syphilis testing. Twenty clinics outside New York City were recruited in 1987 for the blind HIV study. Results for the fourth quarter of 1989 showed seroprevalence rates of 2.8 percent for 2,792 males and 1.1 percent for 1,318 females. The merged data peaked for clients aged 30–39 (4.3 percent). In the racial/ethnic distribution the rate was higher for Hispanics (3.5 percent) than for Blacks (2.5 percent) or Whites (1.7 percent).[3]

Intravenous drug users (IVDU): Blood is routinely collected from clients being admitted to methadone maintenance programs. Four programs with 35 sites in New York City, including all five boroughs, were recruited for the blind HIV study. Clients entering from June 1988 through December 1989 had very high seroprevalence rates (29.9 percent; males, 31.7 percent; females, 26.6 percent). The rates were high for all age groups, all race/ethnic groups, and all boroughs.[3]

Prisoners: All persons entering the state prison system are processed through central intake facilities, where blood specimens are drawn routinely as part of the medical examination. (For female prisoners, see chapter VI.)

In addition to these six windows, seroprevalance data on two other groups ~~were tapped~~: military recruits and first-time blood donors. Additional data sources have been identified, and the Department of Health is considering incorporation of the data into the survey program.

The Centers for Disease Control developed a similar program in late 1987 to survey HIV seroprevalence for six groups: IVDUs, patients at general hospitals, patients at STD clinics, patients at tuberculosis clinics, patients at clinics serving women of reproductive age, and newborns.[4,5] This Family of Surveys is being conducted in more than 30 areas of the United States. New York is one of the participating states in this surveillance system.

Planning and Policy Issues

Legal Basis—In New York State specimens without identifying information can be used for epidemiologic studies without the need for informed consent. New York State Public Health Law (PHL) Article 24-A regulates research on human subjects and requires informed consent of the subject. However, PHL 2441(2) specifically exempts from the definition of human research "the conduct of biological studies exclusively utilizing tissue or fluids after their removal or withdrawal from a human subject in the course of standard medical practice, or . . . epidemiological investigations." Consequently informed consent of the subjects in the seroprevalence study is not required (memo from Peter J. Millock, General Counsel, New York State Department of Health, to Lloyd Novick, August 24, 1987).

Advisory Committees—An Advisory Committee was assembled on September 17, 1987 by the Department of Health to review the objectives and methods of the proposed seroprevalence surveillance system and the newborn study in particular. The members were Elaine Abrams, MD, Harlem Hospital; Daniel Callahan, PhD, The Hastings Center; Victor DeGruttola, MD, Harvard School of Public Health; Richard Kaslow, MD, National Institute of Allergy and Infectious Diseases; Margarite Pappaioanou, MD, Centers for Disease Control; and Warren Winkelstein, Jr., MD, School of Public Health, University of California, Berkeley.

The Advisory Committee unanimously supported the need for the proposed studies and recommended that data be collected by small area and risk behavior to the extent that anonymity was not compromised. Information on prevalence in areas outside New York City was deemed to be highly important for detecting the extent of spread.

A second special Advisory Committee was convened on March 10, 1988, to review the preliminary results of the newborn serosurvey. In addition to Drs. Abrams and Kaslow, the members were

Table I-1—Windows of the New York State HIV Surveillance Project

Seroprevalence Window	Target Population	Sample	Data Collected	Limitations	Implications
Newborns	Childbearing women 275,000 per annum	All births in NYS 275,000 per annum	Age of mother, race/ethnicity of infant, zip code of hospital	Applicability to reproductive age women	a) Childbearing women b) Women of reproductive age c) Pediatric HIV infection
Family Planning Clinics	Women receiving family planning	41 sentinel family planning clinics 13,000 per annum	Age, race/ethnicity, zip code of family planning center	Sentinels are not representative of all women in family planning	a) Women in family planning b) Women of reproductive age
Homeless Adolescents	Homeless and runaway youth	NYC adolescent facility 12,000 per annum	Age, race/ethnicity, gender, risk	Not representative of the adolescent population	a) Document infection in adolescent groups b) Sentinel population to detect changes over time c) Relationship of risk to prevalence of infection
STD Clinics	Clients at county STD clinics	20 STD clinic sites outside of NYC 5,000 per annum	Age, race/ethnicity, sex, zip code of clinic, syphilis	Representative of only those who go to STD clinics	a) Men and women in STD clinics b) Association of HIV-ab and syphilis c) Spread in women, and, by inference, via heterosexual transmission
IVDU Clinics	All IVDU clients of treatment (methadone)	35 methadone maintenance treatment programs & detox programs in NYC	Age, race/ethnicity sex, treatment modality, drugs injected, sexual orientation, needle sharing, treatment (drug) Hx of HIV-related illness	Clients are in methadone-treatment programs (main drug of abuse = heroin, not cocaine). Not representative of addicts not in treatment	a) IVDU newly entering treatment b) IVDU already in treatment
Prisons	NYS prisoners	Sample of inmates entering at the four intake facilities 500 per annum	Prison site, residence (county), sex, age, race, risk group	Applicability to other populations	a) NYS prisoners b) High-risk population (IVDU), minorities c) Fertility information available

Timothy Dondero, Jr., MD, Centers for Disease Control; Myron Essex, DVM, PhD, Harvard School of Public Health; Harvey Fineberg, MD, Harvard School of Public Health; Margaret Heagarty, MD, Harlem Hospital; Keith Krasinski, MD, Bellevue Hospital; Margaret Oxtoby, MD, Centers for Disease Control; Peter Selwyn, MD, Montefiore Medical Center, and Issac Weisfuse, MD, New York City Department of Health.

This Advisory Committee strongly recommended continuance of the newborn serosurvey, particularly for the purpose of monitoring the epidemic.

Laboratory Facilities—A capability to perform the necessary volume of HIV screening and confirmatory tests was developed at the Department's Wadsworth Center for Laboratories and Research. Automation of the testing process, including employment of robotics, facilitated the implementation of a large seroprevalence study. The Wadsworth Center is now performing nearly 400,000 serologic tests for antibody to HIV annually.

Confidentiality—All laboratory HIV determinations are entered into a computerized data-management system. Names of individuals are not collected, and no identifying information is entered into the data system. Demographic data are collected for all studies (age, sex, race/ethnicity, and zip code), and risk factors are collected for some windows (homeless adolescents, drug treatment clients, and prisoners). To prevent potential identification of any individual from demographic information on a single specimen or a small number of specimens, data are reported only on subgroups that include at least six individuals.

Public Information—Extensive public information was provided about the nature and rationale for this effort before inception of the studies. Information on the newborn study was also provided to hospitals and hospital-based pediatricians. Some public concern was initially expressed about the blind nature of these studies and the inability to notify individuals of their results. To allay this concern, voluntary HIV testing and counseling was offered to individuals in most of the participating facilities.

Ethics—Ethical questions have been raised regarding blind surveys, because positive results indicating infection cannot be linked back to the individual for medical follow-up. Under New York State law individuals cannot be notified of test results from nonidentified (blind) samples obtained without consent. The solution taken by the Department of Health was to offer voluntary testing, wherever feasible, to individuals at the same time as the blind testing. Review of the issues raised by blind testing in a number of reports have emphasized the epidemiologic importance of this type of study and its consistency with ethical practice.[7-9]

Data Limitations and Uses—The limitations of data from blind seroprevalence studies were recognized at the outset. The chief limitation is that the groups available for study are determined by the availability of blood specimens. Some population groups, such as adult males, may be difficult to assess, and the study group is not representative of the entire population. There are also limits on the type of information that can be collected, particularly detailed risk histories.

Despite these limitations the serosurvey results are providing critical data on HIV prevalence in the subgroups tested and on changes over time. The newborn study in particular may have wide applicability to the general population of women of reproductive age in the community, since virtually all childbearing women are participants. Issues of completeness and general applicability of the data are dealt with in later chapters.

Collectively these seroprevalence activities are providing invaluable information on the extent of the HIV epidemic in New York State. Information from these studies has directly influenced the development of preventive actions and public policy.

Summary

In the summer of 1987, the New York State Department of Health embarked on an extensive effort to determine the prevalence of trends of HIV infection in the state. Serosurveillance for antibody to HIV was undertaken by blind testing of available blood samples. Six populations were selected as windows on the epidemic: newborns, homeless adolescents, prisoners, and clients of family planning, STD, and drug treatment clinics. Testing of all newborns in New York State began in December 1987, and all six study windows were operational by June 1988. As of March 1990 blind testing for HIV antibody had been performed on more than 725,000 blood specimens in these six studies. Collectively these seroprevalence activities are providing invaluable information and impetus to preventive actions and public policy. ☐

References

1. Centers for Disease Control: HIV/AIDS Surveillance Report, April 1990.
2. New York State Department of Health: AIDS Surveillance Monthly Update. Albany: New York State Department of Health, March 1990.
3. New York State Department of Health: AIDS in New York State. Albany: New York Department of Health, March 1989.
4. Munoz A, Wang M-C, Bass S, *et al:* Acquired immunodeficiency syndrome (AIDS)-free time after human immunodeficiency virus type I (HIV-I) seroconversion in homosexual man. Am J Epidemiol 1989; 130:530–539.
5. Dondero TJ, Pappaioanou M, Curran JW: Monitoring the levels and trends of HIV infection: The Public Health Service's HIV surveillance program. Public Health Rep 1988; 103:213–220.
6. Dondero TJ: Current estimates of HIV prevalence and incidence: Sources and data. Health Policy 1989; 11:115–124.
7. Walters L: Ethical issues in the prevention and treatment of HIV infection and AIDS. Science 1988; 239:597–603.
8. Bayer R, Levine C, Wolf SM: HIV antibody screening: An ethical framework for evaluating proposed programs. JAMA 1986; 256:1768–1774.
9. AIDS and illiberal measures [editorial]. Nature 1987; 325:647.

II. Newborn Seroprevalence Study: Methods and Results

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Introduction

The New York State Department of Health's ongoing study of HIV infection in childbearing women is based on testing of blood specimens obtained from all infants born in this state for seven inherited metabolic disorders. The specimens are collected and initially tested by the Department's Newborn Screening Program. The specimens are then sent to the HIV Seroprevalence Laboratory for blind HIV testing. This paper describes the results for specimens collected during the 28-month period from November 30, 1987 through March 31, 1990.

Since maternal IgG antibodies cross the placenta, the presence of antibodies in a newborn is indicative of infection in the mother. These antibodies do not necessarily indicate that the child is infected. However, an estimated 30–50 percent of seropositive newborns are actually infected.[1-3]

Methods

Testing Protocol

The protocol for HIV testing of newborn blood specimens has been described in detail by Novick, *et al.*[4] Briefly, a dried-blood specimen is obtained from each infant along with certain demographic information, including the mother's age, zip code and county of residence, the newborn's race/ethnicity, and the month and year of birth. Hospitals are informed of the need for accurate, complete reporting and are contacted, if necessary, to obtain missing information. For the HIV study the demographic information—but not identifying information—is entered into a separate, unlinked data base, where each record is identified by a unique, randomly generated number. The same number is assigned to a portion of the blood specimen. All metabolic disorder screening tests are completed before specimens are submitted for HIV testing.

Specimens for which insufficient or unsuitable blood is left after the metabolic disorder screening are excluded from the blinded HIV seroprevalence study. The number of these specimens is carefully tracked. Repeat specimens cannot be obtained at this stage because the blinding process makes it impossible to link the identity of the infant to the specimen. To ensure that only one specimen representing each childbearing woman is tested, the following are also excluded: all specimens except one from a multiple birth event and all repeat specimens requested for follow-up by the Newborn Screening Program (Figure 1).

Repeat Live Births during Study Period

The likelihood that some women would present with a live birth on more than one occasion during the study period was estimated using Vital Records birth files for 1988 and 1989. Although the Vital Records and HIV newborn data are not directly comparable, this method provides a rough estimate of the percentage of women in the HIV newborn study who had two or more separate live birth deliveries during the study period.

Overall, 3.7 percent of the live-birth records indicated a previous live birth within the same two-year period, 1988–89. However, 29 percent of the New York City records and 11 percent of the remaining records were excluded from this analysis because of incomplete or inaccurate information on the date of the last live birth.

Definition of Test Results

Since the initiation of the Newborn Seroprevalence Study, three sets of crite-

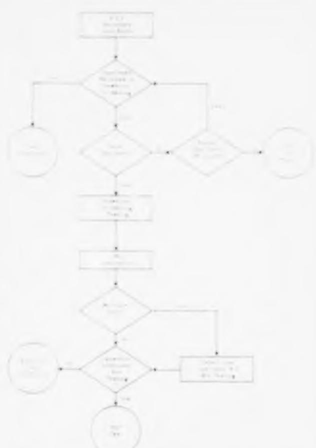

FIGURE 1—New York State Newborn Screening/Newborn HIV Seroprevalence Testing Process

ria have been used to interpret the Western blot results. Reactives were defined as follows:

- Criteria I (November 30, 1987–March 31, 1988): All three of the following: Envelope (gp 41 or gp 120/gp 160), core (p24), and polymerase (p31, p51, or p66).
- Criteria II (April 1, 1988–June 26, 1989): Envelope and either core or polymerase, as defined above.
- Criteria III (June 27, 1989 to present): Any two of the following: p24, gp41, and (gp120 and/or gp160).[5]

Equivocals were defined as virus-specific bands other than the set described above. Nonreactives displayed no virus-specific bands.

Since the individual Western blot band results are currently not available prior to August 1988, the effect of the change from Criteria I to Criteria II could not be examined. However, the change from Criteria II to Criteria III was examined by applying Criteria II to the specimens tested June 27, 1989 to present and comparing the number and percent HIV positive to that found under Criteria III. Under Criteria II there were 1,918 HIV positives with a seroprevalence rate of 0.661 percent. This was nearly identical to that found under Criteria III with 1,921 HIV positives and a seroprevalence rate of 0.662 percent.

Statistical Analysis

Seroprevalence rates were calculated based on the number of Western blot reactives and total specimens tested, excluding equivocals. We could not obtain a second sample for reevaluation of equivocals because of the blinded nature of the study.

Age/race-specific HIV seroprevalence rates were calculated for five racial/ethnic subgroups (White, Black, Hispanic, Other, and Unknown), and six maternal age categories (<20 years, 20–24 years, 25–29 years, 30–34 years, 35+ years and Unknown).

Geographic rates were calculated for New York City total, each of the five New York City boroughs, New York State exclusive of New York City, and four regions outside New York City, which were selected on the basis of urbanization, physical proximity to New York City, and cumulative incidence of AIDS to date. The four regions were New York City Suburban (Nassau, Suffolk, Westchester, and Rockland counties), Mid-Hudson Valley (Dutchess, Putnam, Orange, Sullivan, and Ulster counties), Upstate Urban (Albany, Schenectady, Onondaga, Monroe, and Erie counties), and Upstate Rural (all other counties in the state).

For New York City the HIV seroprevalence rates were also calculated for each zip code area and mapped by quartiles. Zip code areas with fewer than 100 births were eliminated from this mapping. For New York State exclusive of New York City, due to the large number of zip code areas and the small number of births within each area, only the rates for areas with two or more HIV-positive cases and rates twice that of the overall rate were examined, and the rates were mapped by county.

Trend analysis was conducted on the monthly HIV seroprevalence rates for births occurring in the 27-month period, December 1987 through February 1990. Births occurring in November 1987 were excluded from this analysis because the specimens received beginning November 30 covered two-thirds of the November newborns. Plots of the monthly rates were analyzed by simple regression techniques[6–8] with SAS software.[9] A coefficient of determination (R^2) was calculated to determine the proportion of the variation in rates explained by the independent variable, time (month of birth). The 95 percent confidence intervals for the predicted value were also calculated.

This procedure was performed for all of New York State, all of New York City, all of New York State exclusive of New York City, each of the five New York City boroughs, and each of the four other regions described above. The data for all of New York State, all of New York City, and all of New York State exclusive of New York City were further analyzed for three maternal age groups (<20, 20–29, 30+) and for three race/ethnicity groups (White, Black, and Hispanic).

Results

During the study period November 30, 1987 through March 31, 1990, a total of 653,328 blood specimens were analyzed for HIV serologic status, of which 211 equivocals (0.03 percent) were eliminated from the analysis. Of the remaining 653,117 specimens, 10,125 were from women residing outside of New York State, and 2,408 were from women whose residence was unknown. These specimens were also eliminated from the analysis. For the remaining 640,584 specimens, the overall seroprevalence rate was 0.66 percent: 1.24 percent in New York City and 0.17 percent in the rest of the State.

New York City

The HIV seroprevalence rates for New York City and each borough are shown in Table I-1. The rates were highest in the Bronx, followed by Manhattan and then Brooklyn. The rates for Queens and Staten Island were more than twice the rates for the two New York State regions adjacent to New York City, New York City Suburban, and Mid-Hudson Valley.

Of the 170 zip code areas in New York City with 100 or more births, 31 (18.2 percent) had HIV seroprevalence rates in excess of 2 percent and 13 (7.6 percent) had rates in excess of 3 percent. The distribution by quartiles is shown in Figure 2.

The vast majority of HIV seropositives in New York City (89.3 percent) were Black or Hispanic, although only 60.8 percent of newborns were Black or Hispanic.

The highest rates were detected among Black and Hispanic newborns. These rates increased with maternal age for Blacks and Hispanics but not for Whites or Other (Table II-2). The rates for both Black and Hispanic infants peaked in the 30–34 year maternal age group.

Age-specific seroprevalence rates are plotted in Figure 3. The sharpest rise is seen in New York City for infants born to mothers between ages 14 and 24. These rates increased ninefold, from 0.16 percent (1 in 624) for 14-year-olds to 1.41 percent (1 in 71) for 24-year-olds.

TABLE II-1—Percent of HIV Positive Newborns by Region for New York State, November 30, 1987–March 31, 1990

Region	Number Positive	Total Tested	Percent Positive
New York State Exclusive of New York City	601	346,522	0.17
NYC Suburban	329	120,422	0.27
Mid-Hudson Valley	71	29,450	0.24
Upstate Urban	119	88,088	0.14
Upstate Rural	82	108,562	0.08
New York City	3,650	294,062	1.24
Manhattan	799	50,364	1.59
Bronx	998	58,003	1.72
Brooklyn	1,352	104,613	1.29
Queens	424	67,474	0.63
Staten Island	77	13,608	0.57

Women with White seropositive infants were slightly younger (mean 28.6 years) than women with White seronegative infants (29.2 years). Women with Black seropositive infants were significantly older than women with Black seronegative infants (28.2 versus 26.0 years). Women with Hispanic seropositive infants were also significantly older (27.2 versus 25.7 years).

New York State Exclusive of New York City

Seroprevalence rates for New York State and its four regions exclusive of New York City are shown in Table II-1. Rates for two regions adjacent to New York City (New York City Suburban and Mid-Hudson Valley) were only about one-fifth the rate for New York City but twice or three times the rates for the other regions.

HIV seroprevalence was concentrated in certain areas of the state (Figure 4). In New York State exclusive of New York City, 64 zip code areas had two or more seropositives and a HIV seroprevalence rate more than double the average for all areas outside New York City. Of the 64 zip code areas, 50 had rates of 0.5 percent or higher. The 64 zip code areas contained 65 percent of the HIV seropositives but only 16 percent of the newborns tested outside of New York City.

In New York State exclusive of New York City, HIV seroprevalence was highest for Black newborns followed by Hispanics (Table II-3). The majority of HIV seropositives (69.4 percent) were either Black or Hispanic, although only 14.0 percent of the newborns tested were Black or Hispanic. For Black newborns the HIV seroprevalence rate increased with maternal age, peaking in the 35+ age group. For Hispanic newborns the rate increased only in this maternal age group. For White newborns no significant variation with maternal age was found.

As seen in New York City, mothers of White seropositive infants were on average slightly younger than those with White seronegative infants (26.5 years vs 27.7 years); however, the difference is significant in women in New York State exclusive of New York City. Women with Black seropositive infants were significantly older than those with Black seronegative infants (27.7 years vs 24.6 years). Mothers of Hispanic newborns were 1.1 years older (26.5 years vs 25.4 years).

Trend Analysis

When HIV seroprevalence rates were plotted by month of birth for New York City and for New York State exclusive of New York City, with 95 percent confidence intervals, neither slope was significantly different from zero. No significant increase or decrease was found for any borough of New York City, any region outside of New York City, or any three racial/ethnic subgroups in New York State exclusive of New York City.

Only two age groups showed evidence of a statistically significant trend for the 27-month period. For New York City women less than 20 years old (Figure 5A), the rate declined slightly ($y = 0.00785 - 0.000132 \times$, $p < .01$). The standard error of the slope was 0.0000486. However, the coefficient of determination (R^2) was only .23, explaining only a small proportion of the variation in the rate.

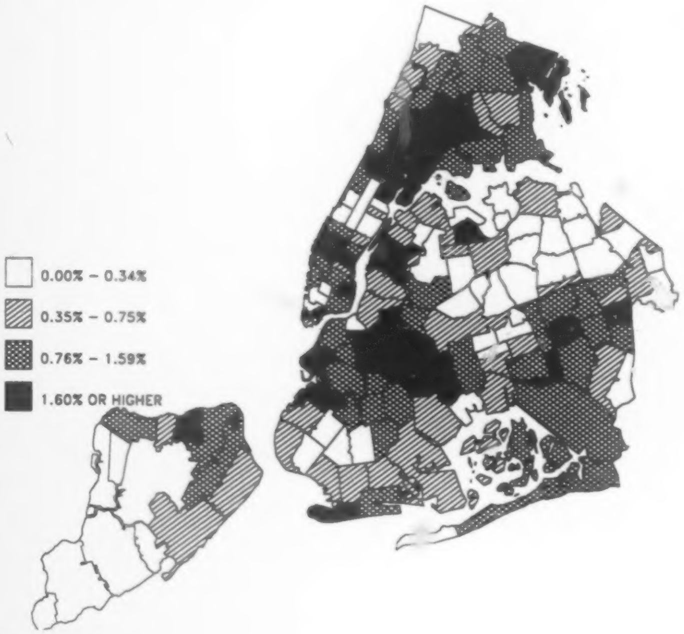

FIGURE 2—HIV Seroprevalence in New York City by Zip Code, November 30, 1987–March 31, 1990

TABLE II-2—Distribution of HIV Positive Newborns by Maternal Age and Race/Ethnicity for New York City, November 30, 1987–March 31, 1990

Race/Ethnicity	<20	20–24	25–29	30–34	35+	Unknown	Total
	HIV Positive by Maternal Age Group (years)						
White							
HIV Positive							
Percent	0.22	0.42	0.38	0.29	0.28	0.74	0.34
Number	6	58	102	74	41	3	284
Tested	2,716	13,935	27,158	25,715	14,403	408	84,335
Black							
HIV Positive							
Percent	0.68	1.68	2.59	3.08	2.98	3.10	2.19
Number	96	429	670	547	268	19	2,028
Tested	14,002	25,512	25,831	17,780	8,985	612	92,722
Hispanic							
HIV Positive							
Percent	0.57	1.26	1.75	1.95	1.42	2.35	1.43
Number	74	333	425	287	101	12	1,232
Tested	12,909	26,519	24,232	14,720	7,113	511	86,004
Other							
HIV Positive							
Percent	0.37	0.43	0.28	0.14	0.23	0.00	0.27
Number	4	23	29	11	8	0	75
Tested	1,095	5,293	10,222	8,037	3,408	147	28,202
Unknown							
HIV Positive							
Percent	0.51	1.10	1.52	0.69	1.76	1.01	1.11
Number	1	5	9	3	4	9	31
Tested	195	454	594	435	227	894	2,799
Total							
HIV Positive							
Percent	0.58	1.18	1.40	1.38	1.24	1.67	1.24
Number	180	848	1,235	922	422	43	3,650
Tested	30,917	71,713	88,037	66,687	34,136	2,572	294,062

For women in New York State exclusive of New York City aged 20–29 (Figure 5B), the rate increased slightly ($y = 0.00149 + 0.00003 \times, p < .03$). The standard error of the slope was 0.0000128. R^2 for this group was extremely small (.17).

Discussion

Before this study was implemented, extremely limited information was available on the prevalence of HIV infection in childbearing women. Two recent studies in New York City hospitals revealed an HIV seroprevalence rate of 2.4 to 2.5 percent in infant cord blood specimens.[10,11] A recent Massachusetts Department of Health study found an overall rate of 0.21 percent in anonymously tested newborn blood samples, ranging from 0.1 percent in suburban and rural hospitals to 0.8 percent in inner-city hospitals.[12]

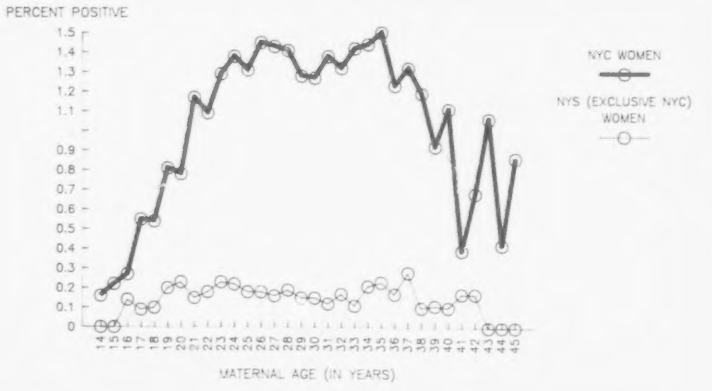

FIGURE 3—HIV Seroprevalence in NYS by Single Year of Maternal Age (November 30, 1987–March 31, 1990)

In this study, we found an overall HIV seroprevalence rate of 0.66 percent for childbearing women. The magnitude of this health problem was most striking in New York City where in some zip code areas as many as one out of 22 childbearing women were found to be HIV-infected. For New York State exclusive of New York City the lower overall seroprevalence rate (0.17 percent) is deceptive, because zip code analysis revealed small areas with rates approaching those in New York City. This small-area information is critical for assessment of large geographic areas, where apparently low seroprevalence may mask communities with rates of serious public health concern.

Although 87 percent of the seropositive newborns in New York State were Black or Hispanic, the seroprevalence rate of 0.12 percent for Whites and for other racial/ethnic groups confirms a significant incursion of HIV infection in those populations as well.

The finding of an increased infection rate with increasing maternal age has implications for the design of preventive activities. An especially sharp rise was seen for adolescents and young women in New York City, from 1 in 624 at age 14 to 1 in 71 at age 24 (Figure 3). Preventive efforts must begin with children and younger adolescents to control this rapid spread.

Although racial/ethnic and maternal-age patterns are crucial for projections and resource allocation, the intersection of these patterns offers deeper insight into the environmental factors that determine prevalence. Identifying such cohorts will help to define the relevant exposures, such as patterns of drug use, and thus the targeting of preventive measures. Individuals in younger cohorts with different exposure risks may exhibit substantially different patterns of HIV infection in the future. Those changes will affect not only projections of the epidemic, but also the measures needed to reduce transmission of the virus.

Certain biases inherent in this study may have an impact on the observed rates. Possible bias resulting from newborns not

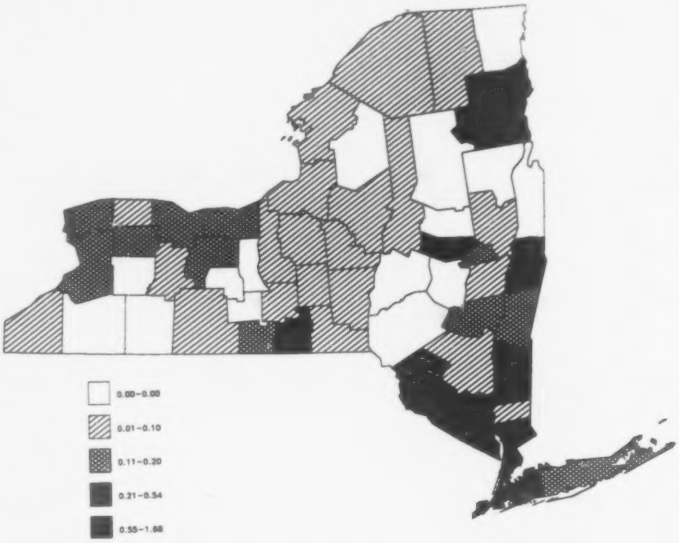

FIGURE 4—HIV Seroprevalence in NYS exclusive of NYC by County (November 30, 1987–March 31, 1990)

TABLE II-3—Distribution of HIV Positive Newborns by Maternal Age and Race/Ethnicity for New York State Exclusive of New York City, November 30, 1987–March 31, 1990

	HIV Positive by Maternal Age Group (years)						
Race/Ethnicity	<20	20–24	25–29	30–34	35+	Un-known	Total
White							
HIV Positive							
Percent	0.05	0.09	0.06	0.04	0.03	0.09	0.06
Number	9	53	57	35	10	1	165
Tested	18,943	59,858	101,038	78,112	29,328	1,095	288,374
Black							
HIV Positive							
Percent	0.26	0.67	1.30	1.60	1.72	1.52	0.95
Number	20	75	120	82	39	2	338
Tested	7,660	11,125	9,245	5,132	2,269	132	35,563
Hispanic							
HIV Positive							
Percent	0.51	0.59	0.55	0.55	1.32	0.00	0.61
Number	10	25	20	11	13	0	79
Tested	1,966	4,243	3,639	1,986	982	51	12,867
Other							
HIV Positive							
Percent	0.00	0.45	0.18	0.13	0.09	0.00	0.19
Number	0	7	5	3	1	0	16
Tested	561	1,563	2,758	2,285	1,058	44	8,269
Unknown							
HIV Positive							
Percent	0.00	0.48	0.00	0.00	0.79	0.20	0.21
Number	0	1	0	0	1	1	3
Tested	56	207	316	246	127	497	1,449
Total							
HIV Positive							
Percent	0.13	0.21	0.17	0.15	0.19	0.22	0.17
Number	39	161	202	131	64	4	601
Tested	29,186	76,996	116,996	87,761	33,764	1,819	346,522

tested is discussed in a later chapter.[13] Another bias could result from double-counting the estimated 3.7 percent of women in the study who may have had two or more separate live birth deliveries within the study period.

Regardless of these biases, data from the seroprevalence study are invaluable in developing estimates of the number of infected women of reproductive age and the number of HIV-infected children. If we assume that 30 percent of seropositive newborns will prove to be infected, 1,275 children with HIV infection were born in New York State during the study period. Yet, only 648 cases of pediatric AIDS had been reported as of March 1990. This gap, attributable mainly to infected but asymptomatic children or those with health problems but no HIV disorder diagnosis, is highly relevant to our understanding of pediatric HIV infection and the need for health care resources.

The few trends found in this study reflect changes in seroprevalence over a brief period of only 27 months. Longer monitoring may be necessary to detect changes in this epidemic. The seroprevalence rates are for a population of childbearing women who have already been infected, plus those infected during the study period, minus those who leave the population by outmigration, death, or aging out of the study. A stable seroprevalence in this limited study may, therefore, be compatible with an actual rising or falling incidence of new infection. The observed trends should also be viewed with caution, because two significant results in 30 separate analyses may be the result of chance alone.

Little information on HIV seroprevalence in newborns is available for the period before late 1987. A rapid increase in HIV infection in childbearing women is assumed to have occurred at some point between 1980 and the present. At the end of 1983, only 174 female AIDS cases had been reported to the New York State AIDS Registry.[14] By the end of March 1990, that number had increased 24-fold to 4,194.[15] The lack of observed trend in the 27-month study period and the increase in infection manifested by AIDS in childbearing women prior to this point suggest a decrease in the rate of increase of the epidemic of HIV infection in childbearing women. However, progress of the epidemic is not likely to be uniform throughout the state with certain lower-prevalence areas possibly exhibiting upward trends in the future.

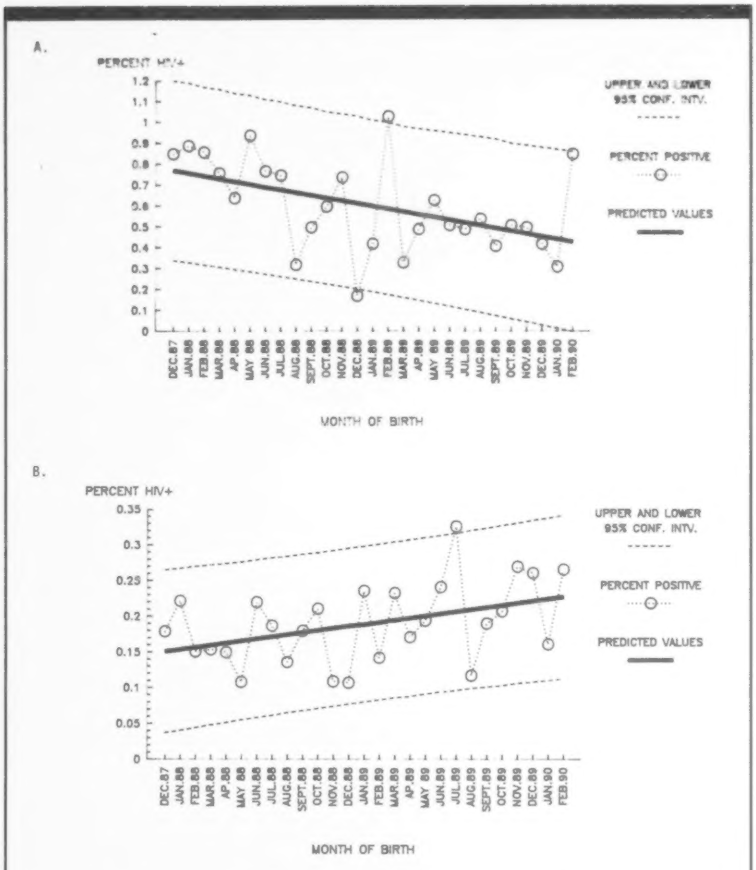

FIGURE 5—Percentage of HIV-Positive Newborns in NYS by Month of Birth for Two Groups with Statistically Significant Trends (December 1987–February 1990)

A—Women less than 20 years old, NYC
B—Women 20–29 years old, NYS exclusive of NYC

Newborn infants of adolescent mothers in New York City showed a decreasing trend in HIV seroprevalence during the 27-month period. This group is particularly critical to the monitoring process as newborns of teenaged mothers have the lowest seroprevalence rates but the greatest rate of increase with maternal age. Seroprevalence among this group may be a closer reflection of recent incidence, since the time interval between HIV infection and childbearing is most likely shorter than that for the older women.

Possible saturation of susceptible individuals in certain geographic areas may also offer an explanation for the lack of observed trends. If so, our efforts toward prevention must be redoubled in these areas, as well as communitywide, to prevent transmission from this pool to others entering various risk groups in the community.

Summary

For the 28-month period, November 30, 1987 through March 31, 1990, 653,117 blood specimens obtained on all newborn infants in New York State for detection of metabolic disorders were also analyzed for HIV serologic status. The overall seroprevalence rate was 0.66 percent: 1.24 percent in New York City and 0.17 percent in New York State exclusive of New York City. Rates of seropositivity were highest in the Bronx (1.72 percent) and Manhattan (1.59 percent). Outside of New York City, HIV seropositivity was concentrated in certain areas. Sixty-four zip codes with two or more seropositives and an HIV seroprevalence rate twice the average outside of New York City contained 65 percent of the HIV seropositives but only 16 percent of the newborns tested. Newborn seropositivity increased with maternal age. In New York City, the seroprevalence rates increased from 0.16 percent (1 in 624) for 14-year-olds to 1.41 percent (1 in 71) for 24-year-olds, a ninefold rise. This survey has provided the impetus for a number of preventive initiatives. ☐

Acknowledgments

Data collection activities were partially supported with funding through cooperative agreements with the Centers for Disease Control.

References

1. The European Collaborative Study: Mother-to-child transmission of HIV infection. Lancet 1988; 2:1039–1043.
2. Blanche S, Rouzioux C, Guihard Moscato M-L, *et al:* A prospective study of infants born to women seropositive for human immunodeficiency virus type 1. N Engl J Med 1989; 320:1643–1648.
3. Thomas PA: The New York City Department of Health: The New York City perinatal HIV transmission study. V International Conference on AIDS, Montreal, Canada, June 8, 1989. Abstract Th.A.0.7.
4. Novick LF, Berns DS, Stricof R, Stevens R, Pass K, Wethers J: HIV seroprevalence in newborns in New York State. JAMA 1989; 261:1745–1750.
5. Centers for Disease Control: Interpretation and use of the Western blot assay for serodiagnosis of human immunodeficiency virus type 1 infections. MMWR 1989; 38(S-7):1–7.
6. Cody RP, Smith JK: Applied Statistics and the SAS Programming Language (second edition). New York: North-Holland, 1987; 70–81.
7. Kleinbaum DG, Kupper LL: Applied Regression Analysis and Other Multivariable Methods. North Scituate, MA: Duxbury Press, 1978.
8. Draper N, Smith H: Applied Regression Analysis, 2d Ed. New York: John Wiley & Sons, 1981.
9. SAS Institute, Inc.: SAS User's Guide: Statistics, Version 5 edition. Cary, NC: SAS Institute, Inc, 1985; 655–709.
10. Landesman S, Minkoff H, Holman S, McCalla S, Sijin O: Serosurvey of human immunodeficiency virus infection in parturients: Implications for human immunodeficiency virus testing programs of pregnant women. JAMA 1987; 258:2701–2703.
11. Krasinski K, Borkowsky W, Bebenroth D, Moore T: Failure of voluntary testing for human immunodeficiency virus to identify infected parturient women in a high-risk population. N Engl J Med 1988; 318:185.
12. Hoff R, Berardi VP, Weiblen BJ, Mahoney-Trout L, Mitchell ML, Grady GF: Seroprevalence of human immunodefi-

ciency virus among childbearing women: Estimating by testing samples of blood from newborns. N Engl J Med 1988; 318:525–530.

13. Pass KA, Schedlbauer LM, MacCubbin PA, Glebatis DM: Comparison of newborn screening records and birth certificates to estimate bias in newborn HIV serosurveys. Am J Public Health 1991; 81(Suppl):22–24, ch. III.
14. New York State Department of Health: AIDS in New York State, through 1989. Albany, NY: New York State Department of Health, April 1990.
15. New York State Department of Health: AIDS Surveillance Monthly Update. Albany, NY: New York State Department of Health, March 1990.

III. Comparison of Newborn Screening Records and Birth Certificates to Estimate Bias in Newborn HIV Serosurveys

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Introduction

For the New York State Newborn HIV Seroprevalence Study of the magnitude of the HIV epidemic among childbearing women, the completeness and validity of the data as representative of the target population are of prime concern. Missed opportunities for HIV antibody testing fall into three categories:

1. The specimen was not received by the Newborn Screening Program;
2. The initial specimen was invalid or unsuitable for testing and a repeat specimen could not be obtained; or
3. The specimen was insufficient or unsuitable for HIV testing after the mandatory testing was completed.

To assess the potential bias from each of these sources, we determined the number and percent of newborns not tested for HIV antibody within each category and compared the distributions of birth site, residence, race or ethnicity, and birthweight for those not tested and those tested.

Methods

To determine the percent of newborns for which an initial specimen was never received (category 1), live-birth records for 1,000 New York State residents were selected from all 1988 vital records by use of a random sample adequate to yield 95 percent confidence intervals with 1 percent error. These records were computer-matched to newborn screening records; for unmatched records, telephone calls were made to the hospital of birth. The number and percent of live-birth records for which no newborn screening record was available were calculated.

For newborn screening specimens initially invalid with no repeat follow-up specimens and for specimens insufficient or unsuitable for HIV testing, the distributions of birth site, residence, race or ethnicity, and low birthweight (less than 2,500 grams) were compared to the distributions for HIV-tested specimens.

To estimate the minimum impact of the bias, the untested specimens in category 2 (invalid-no repeat) and category 3 (unsuitable/insufficient) were grouped by race or ethnicity and region of birth. The two regions were New York City and New York State exclusive of New York City. The observed HIV seropositivity rate for each race/region subgroup was applied to the number of untested specimens in that subgroup. The overall seropositivity rates were then recalculated with the addition of these results.

To estimate the maximum potential impact of bias from all untested newborn, including those in category 1 (never received), an alternative assumption was made that all those untested were HIV-seropositive. The overall rates were then recalculated.

Results

Specimens Not Tested for HIV Antibody

From the 1,000 randomly selected live-birth records, specimens submitted for newborn screening were matched for 985 newborns. For the remaining 15 birth records two specimens were lost in transit, six were never submitted, and seven represented infants born outside New York State. The parents of these seven infants reside in New York State, but no newborn screening specimen would be expected for an out-of-state birth. Thus eight specimens from an expected 993 (0.82 percent) were not received for newborn screening, but 15 of 1,000 specimens

TABLE III-1—Racial/Ethnic Distribution of Newborns in New York State Tested and Not Tested* for HIV by Region of Birth

		Racial/Ethnic Group (%)				
Region and Group	Number	White	Black	Hispanic	Asian	Other/ Unknown
NYC						
Tested	146,000	29	32	28	5	5
Not Tested	2,506	24	38	30	3	5
NYS Exclusive of NYC						
Tested	123,883	84	10	3	1	2
Not Tested	3,392	80	13	4	1	2
Unknown						
Tested	5,747	68	9	7	5	11
Not Tested	393	21	2	1	1	75

*Invalid and no repeat specimen available, or specimen insufficient or unsuitable.
NYC = New York City
NYS = New York State

(1.5 percent) for infants born to New York State residents were not received for HIV testing.

Of the 281,921 initial specimens received in 1988, a total of 275,630 were tested for HIV, while 1,558 (0.5 percent) were invalid and no repeat specimen could be obtained. An additional 4,733 (1.7 percent) specimens had an insufficient quantity left over after mandatory testing or were unsuitable for HIV testing. If the total number of specimens received was increased by 1.5 percent to reflect the estimated number of specimens never received, the expected total of HIV specimens would be 286,150. Of these, 275,630 were actually tested, leaving an estimated 10,520 newborns (3.7 percent) not tested.

Marked differences were found among hospitals in the percentage of newborns not tested (data not shown). Thus the seroprevalence rates were impacted by factors other than the biological transfer from mother to child.

Comparison of HIV-Tested and Not Tested Newborns

For category 2 (invalid-no repeat) and category 3 (specimen insufficient or unsuitable) considered together, 54 percent of untested and 45 percent of tested newborns were from New York City. More untested than tested newborns were associated with an unknown or out-of-state residence (6 percent and 2 percent, respectively).

The racial/ethnic distributions of HIV-tested and not tested newborns by region are presented in Table III-1. In both New York City and New York State exclusive of New York City the untested groups had a higher percentage of Blacks.

Category 2 (invalid specimens with no repeat specimens obtained) had a disproportionate share of specimens at lower birthweight (13 percent, compared to only 9 percent of all specimens received). Birthweight information was not available for 6 percent of the invalid-no repeat group and for only 2 percent of all specimens.

Estimation of Potential Bias

If the assumption is made that untested specimens in categories 2 and 3 had HIV seroprevalence rates equal to those observed for their race/region subgroup, the number of additional HIV-seropositive infants expected would be 54 (Table III-2). Including these untested newborns would increase the overall seroprevalence rate from 0.642 (1,770/275,630) to 0.647 (1,824/281,921).

The estimated number of all newborns not tested for HIV (including category 1) in 1988 is 10,520. Assuming all 10,520 to be seropositive and including them in the calculation would increase the overall seroprevalence rate nearly sevenfold, from 0.64 percent to 4.29 percent.

Discussion

Since our newborn screening program is designed to test all infants born in New York State, use of the dried blood spots for HIV seroprevalence testing ensures that there is no predetermined bias. However, bias may exist due to biologic factors, such as the mother's health or exacerbating medical conditions of the child. This bias may be particularly important in areas with low seroprevalence rates, where a systematic error could distort the calculations.

The results reported here indicate that the seroprevalence study was able to determine the HIV serostatus of all but 3.7 percent of the childbearing women in the state in 1988. Although the proportion not tested is relatively small, it could still be an

TABLE III-2—Estimated Number of HIV+ Newborn among Those Not Tested in Categories Two* and Three**

Region/Race/ Ethnic	Number Not Tested	Percent HIV+	Estimated No. of HIV+
NYC			
White	814	0.34	2.8
Black	1,289	2.17	28.0
Hispanic	1,018	1.43	14.6
Asian	102	0.11	0.1
Other/Unknown	170	0.62	1.1
NYS Exclusive of NYC			
White	2,005	0.06	1.2
Black	326	0.87	2.8
Hispanic	100	0.53	0.5
Asian	25	0.03	0.0
Other/Unknown	50	0.30	0.2
Unknown			
White	82	0.19	0.2
Black	9	4.46	0.4
Hispanic	5	2.22	0.1
Asian	3	0.00	0.0
Other/Unknown	293	0.83	2.4
Total	6,291		54.4

*Initial specimens invalid with no repeat specimens.
**Insufficient or unsuitable specimen for HIV testing.
NYC = New York City
NYS = New York State

important factor if the characteristics of the untested newborns are similar to those population subgroups with high HIV seroprevalence.

Our study indicated that bias may exist from the untested newborns because their racial/ethnic, regional, and birthweight distributions differed from those tested. If these untested newborns were all seropositive, the seroprevalence rate would be increased nearly sevenfold. This is the maximum bias in these data. The bias of 0.005, calculated by applying the existing seroprevalence rates to specimens in categories 2 and 3, is doubtless unrealistically low. The true bias lies somewhere in between.

Initiation of testing newborns for HIV status has focused additional attention on the quality of specimens and completeness of data submitted to the Newborn Screening Program. Several improvements have been observed. At the beginning of the seroprevalence study 12 percent of the specimens submitted were missing key information such as mother's age, county of residence, and ethnicity. Hospitals were called to secure the information. Within four months less than 3 percent of the specimens were submitted with information missing. Similarly, the quality of blood specimens increased due to focused efforts of laboratory staff in dealing with problem facilities.

Summary

Implicit in the New York State Newborn HIV Seroprevalence Study is the assumption that newborns of all New York State residents are tested for human immunodeficiency virus (HIV) antibodies. We examined this assumption by describing that part of the 1988 New York newborn population not tested in the HIV seroprevalence study and assessing any bias contributed by this group. Of the expected total HIV specimens 1.5 percent were never received by the Newborn Screening Program, 0.5 percent were invalid specimens for which no repeat specimen could be obtained, and 1.7 percent were unsuitable or of insufficient quantity to be tested for HIV antibody. Thus 96.3 percent of all 1988 New York newborns were tested for HIV antibody. Black infants from New York City and low-birthweight infants were represented disproportionately among those not tested. Assignment of all untested newborn to HIV-positive status increased the seroprevalence rate 17 percent (0.64 percent to 0.75 percent). ☐

Acknowledgments

The authors thank Joan Cooney and Michael Ellrott of the Bureau of Production Systems Management for supplying data on live births in New York, John Rososki for programming and executing computer programs, and Andrew Stacy for computer graphics.

IV. Geographic Distribution of Newborn HIV Seroprevalence in Relation to Four Sociodemographic Variables

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Introduction

Since the AIDS epidemic was first recognized, there has been geographic clustering of AIDS cases within the United States and New York State. Most of this clustering is related to pools of HIV-infected individuals and to risk behavior activities which occur most frequently in metropolitan areas and states with large populations at risk. Witness the high rates of AIDS cases among homosexual and bisexual men in New York City and San Francisco and among intravenous drug users in urban areas of New York, New Jersey, and Florida.[1,2] In an earlier study in New York City, zip code areas with rates of drug-related hospital discharges in the top quartile had a newborn HIV seroprevalence of 2.2 percent versus 0.8 percent in all other zip code areas.[3]

Identification of geographic clusters of cases can be useful in focusing preventive efforts and allocation of health care resources. HIV/AIDS cases are also likely to be closely clustered in association with other sociodemographic variables. Knowledge of such relationships could be useful for targeting and design of prevention services which would differ between high-risk areas and areas where HIV/AIDS has not yet surfaced.

This report summarizes data on the geographic distribution of HIV seroprevalence among New York State newborns and its association with four sociodemographic variables: birthweight, maternal education, race/ethnicity, and drug abuse leading to hospitalization. Although such associations do not indicate causation, they provide insights for further investigation.

Methods

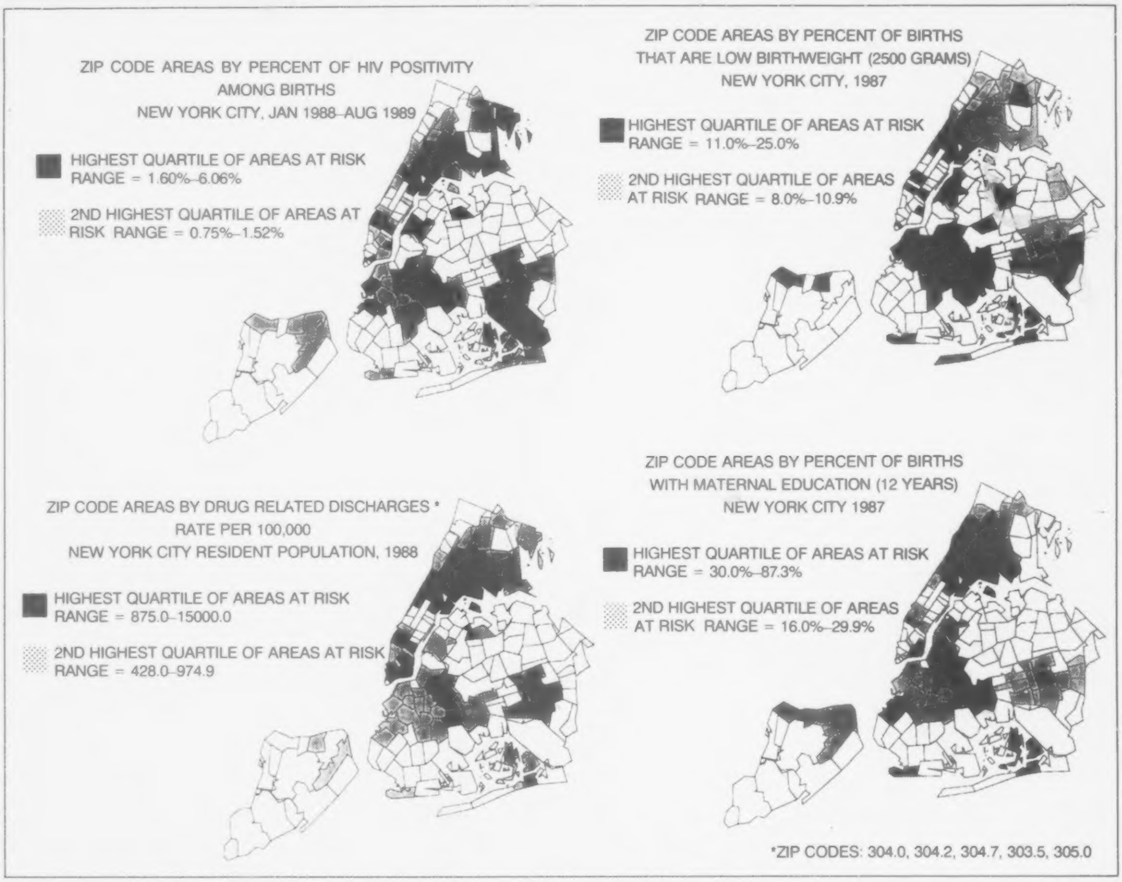
Three sources of data were used: the New York State Newborn HIV Seroprevalence Study, the Birth Certificate Registry, and the New York State hospital discharge file. The data variables analyzed included newborn HIV seroprevalence;[3] hospital discharges with opioid, cocaine, and infant drug withdrawal diagnoses; birthweight; maternal education; and race/ethnicity. All results were converted to rates or percents by zip code, using 1988 postal zip codes.[4]

Newborn HIV seroprevalence and race/ethnicity data included all newborns born after December 31, 1987 and tested for HIV between January 1, 1988 and August 31, 1989. Rates for these variables were calculated as percentages of the total number of newborns tested.

Opioid, cocaine, and infant drug withdrawal diagnoses were obtained from the Statewide Planning and Research Cooperative System for 1988[5] by using the International Classification of Diseases—Ninth Revision codes (304.0, 304.7 and 305.5 for opioid; 304.2 and 305.6 for cocaine; 779.5 and 760.72 for infant drug withdrawal). A combined all-drug variable was established by combining all such adult discharges. Rates per 100,000 population were determined by using zip codes of residence for all persons discharged and zip code population data from the 1985 National Planning Data Corporation estimates.[6] For the infant drug withdrawal rate the denominator was the number of newborn discharges per zip code.

Data on low birthweight (defined as less than 2,500 grams) and low maternal education (defined as fewer than 12 years) were obtained from the registry of 1987 birth certificates.

Geographic comparisons at zip code level were made separately for New York City and for New York State exclusive of New York City. Zip code areas were excluded from the statistical analysis if they had fewer than 100 births, absent variables, or extreme values which were outliers. All variables were mapped by quar-



ZIP CODE AREAS BY PERCENT OF HIV POSITIVITY AMONG BIRTHS NEW YORK CITY, JAN 1988–AUG 1989

■ HIGHEST QUARTILE OF AREAS AT RISK RANGE = 1.60%–6.06%
▨ 2ND HIGHEST QUARTILE OF AREAS AT RISK RANGE = 0.75%–1.52%

ZIP CODE AREAS BY PERCENT OF BIRTHS THAT ARE LOW BIRTHWEIGHT (2500 GRAMS) NEW YORK CITY, 1987

■ HIGHEST QUARTILE OF AREAS AT RISK RANGE = 11.0%–25.0%
▨ 2ND HIGHEST QUARTILE OF AREAS AT RISK RANGE = 8.0%–10.9%

ZIP CODE AREAS BY DRUG RELATED DISCHARGES * RATE PER 100,000 NEW YORK CITY RESIDENT POPULATION, 1988

■ HIGHEST QUARTILE OF AREAS AT RISK RANGE = 875.0–15000.0
▨ 2ND HIGHEST QUARTILE OF AREAS AT RISK RANGE = 428.0–974.9

ZIP CODE AREAS BY PERCENT OF BIRTHS WITH MATERNAL EDUCATION (12 YEARS) NEW YORK CITY 1987

■ HIGHEST QUARTILE OF AREAS AT RISK RANGE = 30.0%–87.3%
▨ 2ND HIGHEST QUARTILE OF AREAS AT RISK RANGE = 16.0%–29.9%

*ZIP CODES: 304.0, 304.2, 304.7, 303.5, 305.0

FIGURE 1—Geographic Comparison of Rates of Newborn HIV Seropositivity, Low Birthweight, Hospital Drug Discharges, and Low Maternal Education by Zip Code, New York City.

tiles for visual ecologic comparison. The geographic distribution of newborn HIV seroprevalence was compared to that of each sociodemographic variable by using Pearson correlation coefficients and multiple regression.

Results

Ecologic Mapping

Visual review of maps of newborn HIV seroprevalence rates by zip code area showed clustering in both New York City (Figure 1) and New York State exclusive of New York City. For New York City all maps of individual drug variables showed a similar pattern so only the most significant variable, all drugs, is presented here. Maps for New York State excluding New York City are not displayed, because release of such data from zip code areas with small numbers could compromise confidentiality.

The first and second quartiles for HIV seroprevalence in New York City had rates of 1.6 to 6.1 percent and 0.7 to 1.5 percent, respectively. The sociodemographic variables were also clustered but appeared to be more closely associated with HIV seroprevalence in New York City than in New York State exclusive of New York City.

Statistical Analysis: New York City

Of the 348 New York City zip code areas listed for 1988, 326 had valid entries listed, but 151 had no population and were excluded, leaving 175. All but five of these areas had a population of 3,798 or more. This restricted file contained 99.5 percent of all births, 99.8 percent of all HIV seropositives, and 99.7 percent of all drug discharges. Further exclusion of nine areas with fewer than 100 births and one area with an extreme drug discharge rate left 165 areas on which the analysis was done. These restrictions entailed minimal loss of data for the variables under consideration (Table IV-1). Over 98 percent of the information for each variable was included in the restricted data set for correlation and regression analyses.

Univariate correlation analysis showed HIV to be correlated in varying degrees with all sociodemographic variables studied in New York City (Table IV-2). Most variables were also highly correlated with one another. If one controlled for low birthweight and the various drug variables in calculating Pearson partial correlation coefficients, low maternal education, percent Black, and percent Hispanic were no longer significantly correlated with HIV. The drug variables were

TABLE IV-1—Number and Percent of Data Points Available for Correlation Analysis by Variable before and after Restrictions, New York City and New York State Exclusive of New York City

Variables	Total Number in Data File	Number Lost To Restrictions*	Number in Analyzed File	Proportional Decrease in Final Analysis
New York City				
Population	7,202,250	37,180	7,165,070	0.5%
Births	196,387	2,732	193,655	1.4%
HIV Positives	2,355	46	2,309	2.0%
All Drug Discharges	47,492	950	46,542	2.0%
Opioid Drug Discharges	28,755	647	28,108	2.3%
Cocaine Drug Discharges	25,701	460	25,241	1.8%
Infant Drug Discharges	4,117	81	4,036	2.0%
New York State Exclusive of New York City				
Population	10,548,363	1,896,471	8,651,892	18.0%
Births	236,648	38,521	198,127	16.3%
HIV Positives	396	54	342	13.6%
All Drug Discharges	12,980	2,179	10,801	16.8%
Opioid Drug Discharges	4,648	877	3,771	18.9%
Cocaine Drug Discharges	9,892	1,575	8,317	15.9%
Infant Drug Discharges	363	40	323	11.0%

*See Text

all highly correlated with one another. A model comprising low birthweight and all drug discharges was shown by multiple regression analysis to be the best predictor of HIV status ($r^2 = .77$). Addition of all remaining variables added little to the model. Low birthweight by itself contributed most to the model ($r^2 = .69$; data available on request).

Statistical Analysis: New York State Exclusive of New York City

Of the 1,918 zip codes listed for New York State exclusive of New York City in 1988, 1,884 had valid entries, but 354 had no population and were excluded. For the remaining 1,530 the median population was 2,441, and 25 percent had populations less than 1,000. This restricted file contained 98.9 percent of all births, 99.2 percent of all HIV seropositives, and 99.2 percent of all drug discharges. Because of small denominators, however, many areas had several extreme or missing values, leading to exclusion of 1,013 areas (1,006 with fewer than 100 births and seven as outliers). These exclusions left only 517 areas for the correlation and regression analyses and resulted in loss of data for a number of variables (Table IV-1). Overall 82 percent of the population, 88 percent of births, 86.4 percent of newborn HIV seropositives, and 83.2 percent of all drug discharges were included in the restricted data set.

Univariate correlation analysis on this restricted data set showed HIV to be less strongly correlated with all variables than in New York City. The variables with the highest correlation were percent Black, percent Hispanic, and the drug variables (Table IV-2). A model comprising percent Black, percent Hispanic, and cocaine drug discharges was shown by multiple regression analysis to be the best predictor of HIV status ($r^2 = .39$). Addition of all remaining variables added little to the model ($r^2 = .43$). The two race variables contributed most to the model ($r^2 = .36$), but all variables were closely associated, and a model without race/ethnicity was almost as good ($r^2 = .42$).

Discussion

The variables used for the analysis were chosen because of their availability, completeness, and suitability as measures of sociodemographic status. Low birthweight has been associated with socioeconomic status.[7] Maternal education, available on the birth certificate, had been demonstrated as a good surrogate for maternal socioeconomic status in previous Departmental studies.[8] Drug discharges, while not a measure of the total drug problem, reflect its distribution by providing a measure of one of its severe outcomes (hospitalization).

Other variables were considered but rejected for one reason or another. For example, sexually transmitted disease rates were not always available by zip code, and Medicaid participation is a biased measure of socioeconomic status because of wide differences in enrollment by locality.

Zip code was chosen as the geographic level of analysis because prevention activities must be targeted to small geographic areas and because a dilutional effect is seen when larger units, such as counties, are used.

Most pediatric AIDS cases are associated with use of intravenous drugs by one or both parents.[1] Of the 590 pediatric cases reported in New York State through October 1989, 346 (59 percent) had mothers who used intravenous drugs, and 108 (18 percent) had mothers who were sexual partners of intravenous drug users.[9] The associations between newborn HIV seroprevalence and hospital intravenous drug discharges in the present study were therefore expected. The high correlation with cocaine discharges, however, is of particular concern. While these mothers may have also used intravenous drugs, the cocaine association could reflect an increased heterosexual transmission of HIV, given the marked increase in syphilis transmission associated with sex in exchange for crack cocaine.[10]

Low birthweight is associated with inadequate health care, poor nutritional status, low socioeconomic status, low level of maternal education, and alcohol and other substance abuse,[7,11-13] including use of cocaine and heroin.[14-16] Thus it was not surprising to find low birthweight closely correlated with newborn seropositivity in New York City. Although drug use, as measured by hospital discharges, added to the association, its contribution was less than that of low birthweight. Hospitalization for drug use measures only the more severe portion of a locality's drug problem. Low birthweight may be a better reflection of the problem for the total community. Theoretically low birthweight could be an outcome of, rather than a surrogate for, HIV seropositivity. However, several studies have shown that there are no significant differences in birthweight between

TABLE IV-2—Correlations between HIV Seropositivity, Low Birthweight, Drug Discharges, Low Maternal Education and Race/Ethnicity, New York City and New York State Exclusive of New York City

	Pearson Correlation Coefficients								
Variables	HIV	LBW	Drug	Opi	Coc	InDr	ME	Blk	His
New York City									
HIV	—	.81	.79	.75	.80	.77	.68	.61	.31
LBW	.81	—	.73	.65	.77	.75	.65	.79	.21
Drug	.79	.73	—	.98	.98	.86	.68	.45	.36
Opi	.75	.65	.98	—	.91	.82	.68	.34	.43
Coc	.80	.77	.98	.91	—	.87	.66	.54	.29
InDr	.77	.75	.86	.82	.87	—	.78	.54	.35
ME	.68	.65	.68	.68	.66	.78	—	.46	.70
Blk	.61	.79	.45	.34	.54	.54	.46	—	−.08
His	.31	.21	.36	.43	.29	.35	.70	−.08	—
NYS Exclusive of NYC									
HIV	—	.33	.49	.41	.50	.35	.33	.50	.43
LBW	.33	—	.44	.33	.46	.35	.52	.55	.23
Drug	.49	.44	—	.84	.98	.37	.44	.67	.47
Opi	.41	.33	.84	—	.72	.23	.31	.44	.46
Coc	.50	.46	.98	.72	—	.41	.50	.70	.45
InDr	.35	.35	.37	.23	.41	—	.30	.34	.21
ME	.33	.52	.44	.31	.50	.30	—	.53	.30
Blk	.50	.55	.67	.44	.70	.34	.53	—	.30
His	.43	.23	.47	.46	.45	.21	.30	.30	—

LBW = Low Birthweight
Drug = All Drug Discharges
Opi = Opioid Discharges
Coc = Cocaine Discharges
InDr = Infant Drug Discharges
ME = Maternal Education < 12 Years
Blk = Percent Black
His = Percent Hispanic

newborns of HIV-positive versus HIV-negative women when the analysis controls for drug use and socioeconomic status.[17-19]

Race/ethnicity was correlated with HIV seroprevalence in New York City, but this correlation appeared to be related primarily to low birthweight and drug use. This finding parallels AIDS case rates, where minorities have much higher case rates but primarily as a function of intravenous drug use.[20]

A different pattern of correlations was seen in New York State exclusive of New York City. The geographic distributions of all sociodemographic variables were still correlated with newborn HIV seroprevalence by zip code, but the correlation coefficients were far less strong. Also, on multiple regression analysis, race/ethnicity replaced low birthweight as the most significant, albeit minimal, predictor of HIV status, while drug use remained a contributor.

Minorities have previously been shown to be disproportionately affected by AIDS, with case rates three to 12 times those of Whites.[21] Blacks and Hispanics contribute 24 percent and 14 percent of the United States AIDS cases, respectively, while comprising only 12 percent and 7 percent of the population.[22] Among AIDS patients, 71 percent of all women are Black or Hispanic, as are 77 percent of all children.[1] These differences have been repeatedly associated with intravenous drug use and secondary heterosexual and perinatal transmission.[20-23]

The reason why race/ethnicity appears to be a better correlate for HIV than low birthweight or drug use in New York State exclusive of New York City is unclear. Some possible explanations are the smaller amount of data available for analysis by zip code, the lower prevalence of drug use resulting in hospitalization (drug discharges per 100,000 population were 17 percent of those in New York City), and the lower rate of newborn HIV seroprevalence (0.16 versus 1.25 percent). Furthermore, the ratios of AIDS case rates for Blacks and Hispanics compared to Whites have been much greater in New York State exclusive of New York City (7.6 and 5.2, respectively, versus 2.1 and 2.0). Similarly, the ratios of HIV newborn seroprevalence rates for Blacks and Hispanics are 15 and 10 times higher, respectively, than for Whites in New York State exclusive of New York City, compared to six and four times higher in New York City.

While the major emphasis for public health HIV/AIDS programs has been on universal education and precautions, efforts targeted at high-risk persons or geographic areas are also important. Our results confirm that prevention activities in New York City should be focused on geographic areas that have low HIV/AIDS rates but populations with low birthweights and high drug use—that is, populations at risk for future HIV/AIDS activity—as well as on geographic areas with already high HIV/AIDS rates. The extent to which sociodemographic factors are important predictors outside New York City remains to be seen.

Summary

The geographic distribution of newborn human immunodeficiency virus seroprevalence at zip code level was compared with the distribution of four sociodemographic variables. For New York City significant univariate correlations were found between HIV and low birthweight, drug use (as measured by hospital discharges), maternal education less than 12 years, and race/ethnicity. Less significant correlations were found for New York State exclusive of New York City. For New York City a model comprising low birthweight and all drug discharges was shown by multiple regression analysis to be most strongly associated with HIV status ($r^2 = .77$). Elsewhere a model comprising race/ethnicity (percent Black, percent Hispanic) and cocaine

drug discharges had the best, but less strong association ($r^2 = .39$). However, because of intercorrelations, race/ethnicity added little when the other variables were included first. Knowledge of the geographic association between HIV seroprevalence and sociodemographic status can be useful in designing and focusing prevention efforts in areas at highest risk for future HIV/AIDS activity. □

Acknowledgments

The authors are indebted to Joanne Sikorski, Michael Davisson, William Wallace, Hwa-Gan Chang, and Rachel Stricof of the New York State Department of Health for their assistance in preparation of the manuscript.

References

1. Centers for Disease Control: HIV/AIDS Surveillance Report. Atlanta, GA: Centers for Disease Control, October 1989; 1–16.
2. New York State Department of Health: AIDS in New York State. Albany, NY: New York State Department of Health, March 1989.
3. Novick LF, Berns DS, Stricof RL, Stevens RW, Pass KA, Wethers JA: HIV seroprevalence in newborns in New York State. JAMA 1989; 261:1745–1750.
4. 1988–1989 five digit zip code boundary file. Lyme, NH: Geographic Data Technology, Inc., 1989.
5. New York State Department of Health: New York State Hospital Discharges. Statewide Planning and Research Cooperative System. Albany, NY: New York State Department of Health, 1988.
6. 1985 population estimates by zip code. Ithaca, NY: National Planning Data Corporation, 1985.
7. Committee to Study the Prevention of Low Birthweight, Division of Health Promotion and Disease Prevention, Institute of Medicine: Preventing Low Birthweight. Washington, DC: National Academy Press, 1985.
8. Therriault G: Development of socioeconomic measures in the analysis of vital statistics data. Presented at the Public Health Conference on Records and Statistics, Washington, DC, June 1978, sponsored by the National Center for Health Statistics.
9. New York State Department of Health: AIDS Surveillance Monthly Update. Albany, NY: New York State Department of Health, October, 1989; 1–34.
10. Centers for Disease Control: Congenital Syphilis—New York City, 1986–1988. MMWR 1989; 38:825–829.
11. Behrman RE: Preventing low birth weight: A pediatric perspective. J Pediatr 1985; 107:842–854.
12. Miller AC, Jekel JF: Incidence of low birth weight infants born to mothers with multiple risk factors. Yale J Biol Med 1987; 60:397–404.
13. McAnarney ER: Young maternal age and adverse neonatal outcome. Am J Dis Child 1987; 141:1053–1059.
14. Gregg GE, Davidson DG, Weindling AM: Inhaling heroin during pregnancy: Effects on the baby. Br Med J 1988; 296:754.
15. Chasnoff IJ, Lewis DE, Griffith DR, Wiley S: Cocaine and pregnancy: Clinical and toxicological implications for the neonate. Clin Chem 1989; 35:1276–1278.
16. Keith LG, MacGregor S, Friedell S, Rosner M, Chasnoff IJ, Sciarra JJ: Substance abuse in pregnant women: Recent experience at the Perinatal Center for Chemical Dependence of Northwestern Memorial Hospital. Obstet Gynecol 1989; 73:715–720.
17. The European Collaborative Study: Mother-to-child transmission of HIV infection. Lancet 1988; 2:1039–1043.
18. Selwyn PA, Schoenbaum EE, Davenny K, Robertson VJ, Feingold AR, Shulman JF: Prospective study of human immunodeficiency virus infection and pregnancy outcomes in the intravenous drug users. JAMA 1989; 261:1289–1294.
19. Johnstone FD, MacCallum L, Brettle R, Inglis JM, Peutherer JF: Does infection with HIV affect the outcome of pregnancy? Br Med J 1988; 296:467.
20. Selik RM, Castro KG, Pappaioanou M: Distribution of AIDS cases, by racial/ethnicity group and exposure category, United States, June 1, 1981–July 4, 1988. MMWR 1988;37 (Suppl. No. SS-3):1–3.
21. Curran JW, Jaffe HW, Hardy AM, Morgan WM, Selik RM, Dondero TJ: Epidemiology of HIV infection and AIDS in the United States. Science 1988; 239:610–616.
22. Hopkins DR: AIDS in the minority populations in the United States. Public Health Rep 1987; 102:677–681.
23. Bakeman R, Lumb JR: AIDS statistics and the risk for minorities. AIDS Res 1986; 2:249–252.

V. Projection of AIDS Incidence in Women in New York State

Lawrence Lessner, PhD, MPH

Introduction

One objective of the New York State Newborn HIV Seroprevalence Study was to provide information for AIDS case projections. Earlier projections were developed by linear extrapolation from reported AIDS cases. The limitations of using AIDS case reports include underreporting of AIDS cases, reporting delays, and, most importantly, the long incubation period of HIV infection.

To improve the projection process, we turned to data from two other sources: fertility data, and the results of the Newborn HIV Seroprevalence Study. With these and other data, we estimated the number of HIV-infected women of reproductive age in 1988 and then the number of women infected each year during 1981–88. From this distribution of the incidence of infection and an incubation distribution, we projected the anticipated AIDS case incidence for this population.

The AIDS case projection methods developed here are a form of back calculation.[1,2] The major advantage of this application is that information from another data source, newborn HIV seroprevalence, is employed to obtain an estimate of the number of infected women of childbearing age in 1988.

As newborn serosurveys for antibody to HIV are implemented in more states, this type of analysis will become increasingly important in efforts to anticipate future AIDS cases.

Methods

AIDS case projections were developed in three steps:

1. Newborn seroprevalence results from 1988 provided an estimate of the number of infected women of reproductive age in 1988.

2. An incubation distribution was used to create an expected case series for each number of a family of hypothetical distributions of incident infections. A "best distribution" of infections was selected by comparing the expected and actual case series.

3. A lower bound on future AIDS cases for this population was obtained from the estimates of presently infected women.

Estimating the Number of Infected Women

The number of HIV-seropositive newborns in 1988 was obtained from the Newborn HIV Seroprevalence Study[3] for each of 80 age, racial/ethnic, and geographic strata. The number of HIV-infected women of reproductive age (15–44 years) in each stratum was determined by dividing the number of newborn HIV seropositives[3] by the fertility rate for that stratum. The total estimate is the sum of these 80 stratum-specific estimates.

The fertility rate R of a given female population is defined as B, the number of live births that occur in the population during the given year, divided by P, the average or midyear population for the given female subgroup of age 15 to 44 years: $R = B/P$. An estimate of P_i, the size of the infected female population, was obtained by inverting the relationship: $R_i = B_i/P_i$. If B_i is the number of seropositive births and R_i is the fertility rate in the infected female population, the population size is $P_i = B_i/R_i$. Assuming the same fertility rate for infected women as for the entire stratum $R_i = R$, then $P_i = B_i/R$.

Since the fertility rate R varies by age, race, and geographic location, the population was stratified by age (15–19, 20–29, 30–39, 40–44 years), race (Hispanic, non-Hispanic Black, non-Hispanic White, and all others), and five geographic regions: New York City (the five bor-

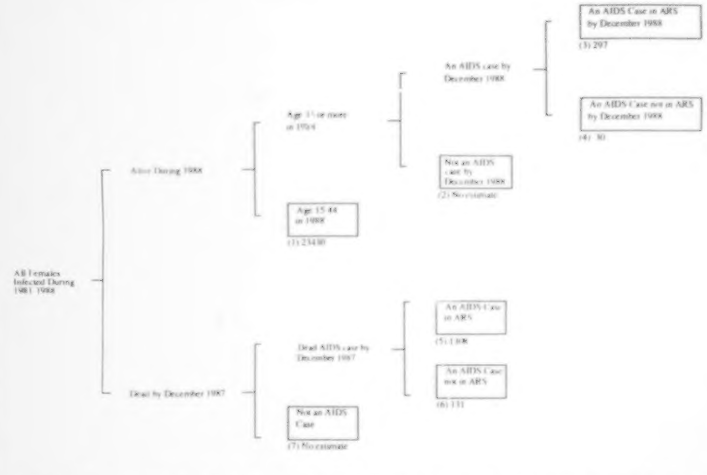
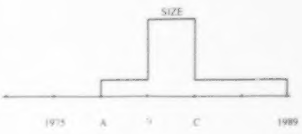

All Females Infected During 1981-1988
Alive During 1988
Dead by December 1987
Age 45 or more in 1988
Age 15-44 in 1988
(1) 23430
An AIDS case by December 1988
Not an AIDS case by December 1988
(2) No estimate
An AIDS Case in ARS by December 1988
(3) 297
An AIDS Case not in ARS by December 1988
(4) 30
Dead AIDS case by December 1987
Not an AIDS Case
(7) No estimate
An AIDS Case in ARS
(5) 1308
An AIDS Case not in ARS
(6) 131

FIGURE 1—Procedure for Estimating the Total Number of Women Infected During 1981–88



A The beginning of the infection epidemic curve, called START, has values 1975, 76, 77, 78, 79, 80

B Called CENTER, is the beginning of the period of high injection activity and has values 1975 to 1980 and is constrained by A≤B

B,C The interval from B to C is called the period of high infection activity, has length C - B = WIDE, has values 1, 2, 3, 4 years

SIZE Is the proportion of cases in the period of high infection activity, has values .5, .6, .7, .8, and .9

FIGURE 2—Generic Candidate For Distribution of Infections

oughs); New York City Vicinity (Nassau, Suffolk, and Westchester Counties), Mid-Hudson Valley (Dutchess, Orange, Putnam, Rockland, Sullivan, and Ulster Counties), Upstate Urban (Albany, Schenectady, Onondaga, Erie, and Monroe Counties), and Upstate Rural (all other counties).

Vital statistics data for 1980 and 1988 were used to obtain estimated stratum-specific fertility rates. The 1980 fertility rates were increased in each stratum to reflect the 1988 fertility experience: ages 15–19 were increased by 17.8 percent, ages 20–29 by 4.1 percent, ages 30–39 by 30.5, and ages 40–44 by 48.9 percent (J. Cooney, Bureau of Production Systems Management, New York State Department of Health, oral communication, January 1990).

The model for estimating the number of women infected during 1981–88 is presented in Figure 1. This tree diagram partitions the ever-infected population into disjoint subgroups for which infection is estimated. Subgroups 4 and 6 use estimates from the AIDS Reporting System (ARS) which is considered 80 to 90 percent complete (Dr. Meade Morgan, Chief of Statistical and Data Management Branch, Division of HIV/AIDS, Centers for Disease Control, oral communication, January 19, 1990).

Estimating the Distribution of Infections

A family of candidate distributions was examined for their utility in describing the incidence of HIV infection in women during 1981–88. The generic candidate (Figure 2), a two-step histogram, was generated by specifying four parameters: START, WIDE, CENTER, and SIZE. START, the beginning of the infection epidemic, had possible values 1975, 1976, . . ., 1980. The interval of high infection activity begins at CENTER and had possible values that range from START to 1988. WIDE is the width of the period of high infection activity and had possible values 1, 2, 3, or 4 years. SIZE is the proportion of infections within the period of intense infection activity and had possible values 0.5, 0.6, 0.7, 0.8 or 0.9. With this model there are 1,200 possible two-step histograms.

A Weibull incubation distribution was selected with a median of 10 years and a fifth percentile of 3.5 years. This selection was based on reports of incubation distributions in a hemophiliac population and in the San Francisco Gay Cohort,[4,5] with estimated median incubation times of 10.0 and 9.8 years, respectively. The estimated fifth percentiles of incubation times were 3.6 and 3.4 years, respectively. An expected case series was created for each of the 1,200 candidates and compared to the annual observed, lag-adjusted AIDS case incidence.

The comparison measure, called ERROR, is the sum of the absolute differences between the observed and expected incidences for each year in each candidate series. The 1,200 ERROR sums were ordered from smallest to largest. Absolute accuracy was measured by ERROR. Relative accuracy was obtained with %ERROR, which is ERROR divided by 2,589, the total incidence observed during 1981–88.

To validate the use of an incubation distribution with a median of 10 years and a fifth percentile of 3.5 years, a family of nine Weibull incubation distributions was created having a 50th percentile of 9, 10, or 11 years and a 5th percentile of 3.0, 3.5 or 4.0 years. Each of these incubation distributions and each of the 1,200 histograms of infection were combined to produce 10,800 candidates, and for each candidate an expected case series was calculated. The top 50 candidates, as ranked by ERROR, were examined to find the smallest ERROR for each incubation distribution (Table V-1). This calculation was at least consistent with the choice of 10 and 3.5 years, although the choices (10, 4.0) and (9, 4.0) appeared equally good. The choices where the 50th percentile is 11 or the 5th percentile is 3.0 appeared considerably less consistent with the observed case series.

Projections of AIDS Case Incidence in Women

The candidate with the minimum value of ERROR was selected as the estimate of the distribution of infections. The expected number of cases was calculated for each year as a lower bound on the anticipated future incidence of AIDS cases in women.

TABLE V-1—Comparison of Incubation Distribution Parameters and ERROR of the Best Candidate

Candidates for the 50th Percentile	Candidates for 5th Percentile		
	3.0	3.5	4.0
9	(*)	70.9	58.9
10	123.7	61.0	61.9
11	121.4	91.2	105.8

(*) at least 154.5

NOTE: Entries are the ERROR for the best candidate for each combination of parameters

TABLE V-2—Summing the Decision Tree (Figure 1)

Box	Population (Origin of the estimate)	The Estimate
1	Women of childbearing age in 1988 (Inverting the fertility rates over 80 strata)	23,430
2	Alive in 1988 and age 45 or more, but not an AIDS case by Dec. 1988 (Unknown)	No estimate
3	An AIDS case in ARS by Dec. 1988 (ARS)	297
4	An AIDS case by Dec. 1988 but not in ARS (10% of 297)	30
5	AIDS case in ARS and dead by 1987 (ARS)	1,308
6	Dead AIDS case by Dec. 1987 and *not* in ARS (10% of 1,308)	131
7	Not AIDS case and died from either other causes or HIV-related disease not AIDS (Unknown)	No estimate
Total		25,196

Results

Estimates of Number of Infected Women (15–44 years)

If we assume equal fertility rates for the general and infected populations in each of 80 age, racial/ethnic, and geographic strata, an estimated total of 23,430 women age 15–44 years were infected with HIV in New York State in 1988. This total was used to obtain an estimate of all women infected during 1981–88 (Figure 1, Table V-2).

Estimates were not possible for certain populations. Box 2 represents all women infected during this period who were alive and age 45 years or more in 1988 but who were not a defined Centers for Disease Control (CDC) case by December 1988. This is the only source of infected women who are potential future AIDS cases for which there is no estimate. Box 7 represents all women infected during this period who died by December 1987 from causes not related to HIV infection. This population will not influence future AIDS incidence.

The estimate of all HIV-infected women of reproductive age in New York State during 1981–88 is 25,196.

Estimates of Distribution of Infections during 1981–88

The 1,200 candidates were ordered by increasing values of ERROR, and the observed and expected cases for six representative candidates were compared (Table V-3). The best candidate had half the ERROR of the next-best candidate.

Three populations of candidates for the infection distribution—the best 10, 20, and 30 candidates—were examined for consensus on the four parameters (Table V-4). START had a clear single mode across all three populations. This is a strong consensus that year 1980 was the beginning of the infection distribution. For CENTER there was a strong consensus across all three populations for 1984 and 1985.

The consensus for the percentiles (Table V-5) shows that the cumulative proportion of new infections nearly doubles in 1984 for each of the three populations. Thus the period of intense infection activity began in 1984. The three-year period 1984–86 accounts for 70 to 80 percent of all infections.

Projections of Future AIDS Cases

The three best candidates for the distribution of infections were used with the incubation distribution to project future AIDS case incidence for 1989 to 1993 (Table V-6). A considerable consensus was found for each year across the three best candidates, and the means and standard deviations for the top 20 candidates substantiated this consensus. The average observed coefficient of variation for these five years was 0.0313. The best candidate indicates that if there were no additional infections after December 1988, the minimum cumulative number of cases expected December 1993 is 8,295.

The linear projections in Table 6 are estimates of future AIDS incidence calculated from actual monthly AIDS surveillance data by linear extrapolation.[6] These linear projections and the best candidate show considerable similarity for the first three years. Beginning in 1992 there is a growing divergence. The linear projections continue to increase each year by a fixed amount, 277 cases. The rate of increase in the candidates is governed by the distribution of infections and, in time, if one assumes no additional infections, there will be a slowing in the rate. Since these projections make no attempt to anticipate future infections, they must be viewed as a conservative estimate.

Discussion

These projections of future AIDS cases among women of reproductive age are based on the results of a newborn HIV seroprevalence study in New York State. An earlier method, linear extrapolation, yielded similar projections for the short term.[6] Projections by extrapolation will always exceed those produced with a method that uses a fixed number of infected because the projections of AIDS cases produced here are based only upon presently infected persons; they must be considered a lower bound of expected cases.

Back calculation is a general class of methods that uses an incubation distribution and the observed case incidence series to estimate the distribution of infections over time.[1,2] The method discussed here, employing two-step histogram, suffers from all the usual disadvantages of back-calculation methods: underreporting of case incidence, limited knowledge of the incubation distribution, and absence of case reports before a certain year (in this case 1981). The key theoretical advantage of back calculation, compared to earlier empiric extrapolation, is its direct quantification of the size of a previously infected population.

The potential limitations of the validity of the work presented here include the assumption of equal fertility between the general and the infected populations in each stratum, the selection of an incubation distribution, the simplicity of the two-

TABLE V-3—Expected Case Series for Selected Best Candidates, Assuming 25,196 Total Infected Woman and a Weibull Incubation Distribution Having 50th Percentile of 10 Years and 5th Percentile of 3.5 Years

Year	Observed	Best	Best 2nd	Best 3rd	Best 10th	Best 20th	Best 30th
1981	9	18	29	32	19	23	21
1982	35	50	58	65	52	62	58
1983	96	99	98	110	104	124	116
1984	155	168	159	168	183	210	196
1985	267	269	262	253	305	319	298
1986	435	436	420	411	476	458	426
1987	658	670	637	644	697	649	595
1988	934	939	901	915	957	889	810
Total	2589	2649	2564	2598	2793	2734	2520
ERROR		61.04	121.8	152.07	204.41	253.97	323.23
%ERROR		2.40	4.7	5.90	7.90	9.80	12.50

step histograms, and the underrepresentation of infections occurring during the survey period, 1988.

A fundamental assumption in relating newborn seropositivity to infections in all women of reproductive age is that HIV-infected women have the same fertility rate as uninfected women in the same age, racial/ethnic, and geographic strata. However, the possible effects of HIV infection challenge this assumption. Intravenous drug use could also reduce fertility and thus low-bias the estimate of infected women of childbearing years.

Few published reports address the potential differences in fertility between the general population and HIV-infected or intravenous-drug-using women. Commonly the comparison is between seropositive and seronegative women who are also or have been intravenous drug users.[7-10] For these groups no differences in fertility have been observed.

If there is a decrease in fertility in HIV-infected women, it may be secondary to associated intravenous drug use. Selwyn[7] compared the pregnancy rate of women in the Bronx in 1985 with a cohort of intravenous drug users enrolled in a methadone maintenance treatment program. The latter group had an estimated decrease in the fertility rate of 4.7 pregnancies per 100 person-years. Thus if there is a decrease in fertility, it is likely to be small (Dr. Peter Selwyn, Department of Epidemiology and Social Medicine, Montefiore Medical Center, New York, NY, oral communication, January 1990).

To investigate the sensitivity of the assumption of equal fertility, we computed the estimate of total infected women for progressively decreasing fertility rates in the infected population. If

$$\sum_{i=1}^{80} \frac{B_i}{R_i}$$

is the original estimate of the total number of infected women of childbearing age in 1988,

$$\sum_{i=1}^{80} \left[\frac{B_i}{(1-a)R_i} \right] = \left[\frac{1}{1-a} \right] \sum_{i=1}^{80} \frac{B_i}{R_i}$$

is the estimate resulting from a 100a percent decrease in fertility. The results of these calculations are given in Table V-7, as well as the ERROR for the best, the best five, and the best 10 candidates for each scenario of decreased fertility. These ERROR values measure the consistency of the assumed decrease in fertility.

As fertility decreases, the estimated total number of infected women increases to 48,626 (corresponding to a 50 percent decrease in fertility). The three ERROR values generally increase with decreases in fertility. This generally increasing ERROR implies a degradation in the consistency of the model and casts doubt on the estimated total number of infected women that results from a severe decrease in fertility. Modest decreases in fertility result in larger but still credible estimates of infecteds (a 15 percent decrease in fertility results in a 16 percent increase in infecteds and a 13 percent increase in best-10 ERROR). Larger decreases in fertility are clearly much less credible than the estimate of no difference in fertility rates between the HIV-infecteds and the general population.

The selection of the incubation distribution was based upon two articles[4,5] that indicated consensus in the choice of a Weibull distribution and its parameters.

TABLE V-4—Consensus for Distributional Parameters in the Best 10, 20, and 30 Candidates

Variables	Best 10	Best 20	Best 30
START			
75	1	1	1
76	0	1	1
77	0	2	2
78	0	2	5
79	3	4	7
80	6	10	14
SIZE			
0.5	1	2	2
0.6	3	3	5
0.7	2	5	10
0.8	3	7	9
0.9	1	3	4
WIDE			
1	2	6	8
2	2	3	5
3	4	7	12
4	2	4	5
CENTER			
84	5	10	12
85	4	8	14
86	1	2	4

TABLE V-5—Consensus for Percentiles of Incident Infections in the Best 10, 20, and 30 Candidates

Year	Best 10	Best 20	Best 30
1980	.06 (.01)	.06 (.01)	.06 (.02)
1981	.10 (.02)	.10 (.03)	.10 (.03)
1982	.15 (.04)	.13 (.04)	.14 (.04)
1983	.19 (.06)	.17 (.06)	.18 (.06)
1984	.34 (.07)	.32 (.09)	.30 (.09)
1985	.61 (.12)	.67 (.16)	.63 (.17)
1986	.87 (.09)	.88 (.10)	.86 (.11)

NOTE: The average cumulative number of new infections from 1975 to the given year for the Best 10, 20 and 30 Candidates is shown. The standard deviation for the data is given in parentheses

Attempts at verification were consistent with those reported in both articles.

Although structurally more complex histograms would yield more accurate results, our simple histograms still offer a wide variety of choices. In any case, parametric modelers performing a back calculation with maximum likelihood methods must necessarily limit the form of their candidate populations for each estimate.[2] The accuracy of the expected case series in the best several candidates (Table V-3), the strength of consensus in parameter estimates (Table V-4), and the strength of consensus in the future num-

TABLE V-6—Projections of Future AIDS Cases from Seroprevalence Data and by Linear Extrapolation

Year	Linear Projections*	Seroprevalence Projections				
					Summary of the Top 20 Candidates	
		Best	2nd Best	3rd Best	Mean	SD
1989	1170	1221	1180	1199	1232	47.5
1990	1446	1487	1447	1469	1502	53.0
1991	1723	1714	1679	1702	1732	54.9
1992	1999	1885	1858	1880	1904	52.7
1993	2276	1988	1970	1989	2006	46.6
Total 1989–93	8614	8295	8134	8239	8376	

*Estimates of future AIDS incidence from an empiric model using actual monthly AIDS surveillance data.

TABLE V-7—Sensitivity and Consistency of the Estimate of the Total Number of Women Infected during 1981 to 1988 to Decreases in the Fertility Rate of Infected Women

% Decrease in Fertility of Infected Women	# Women of Childbearing Years	Total Number Infected during 1981–1988	ERROR		
			Best	Best 5	Best 10
0	23430	25196	61	134	164
10	26033	27799	107	144	190
12	26565	28331	71	140	180
15	27565	29331	94	151	185
17	28118	29884	125	162	195
20	29288	31054	175	209	229
22	29897	31663	176	222	237
30	33472	35238	88	175	242
42	40217	41983	206	246	289
50	46860	48626	254	328	363

bers (Table V-6) confirm the adequacy of the simple histograms.

This method, utilizing HIV seroprevalence data from newborns, has resulted in improved projections of AIDS case incidence in women and may be useful in developing improved projections of pediatric AIDS.

Summary

The results of the Newborn HIV Seroprevalence Study in New York State for 1988 were used to estimate the number of human immunodeficiency virus (HIV)-infected women of childbearing age in 1988. The estimate was accomplished for each of 80 age, racial/ethnic, and geographic strata by dividing the number of seropositive newborns by the 1988 estimated fertility rate. Summing across strata yielded a total of 23,430 infected women age 15 to 44 years in 1988. From this estimate a tree model was used to estimate the number of women infected during 1981–88. Back calculation was used to distribute these infections over time, and the selected incubation distribution was used to estimate future AIDS cases for this population: 8,295 cases by 1993. ☐

References

1. Brookmeyer R, Gail MH: Methods for projecting the course of the AIDS epidemic. JNCI 1988; 80:900–911.
2. Brookmeyer R, Damiano A: Statistical methods for short-term projections of AIDS incidence. Stat Med 1989; 8:23–34.
3. Novick LF, Glebatis DM, Stricof RL, MacCubbin PA, Lessner L, Berns D: Newborn seroprevalence study: Methods and results. Am J Public Health 1991; 81(suppl): 15–21, Ch. II.
4. Brookmeyer R, Goedest JJ: Censoring in an epidemic with an application to hemophilia-associated AIDS. Biometrics 1989; 45:325–335.
5. Bacchetti P, Moss AR: Incubation period of AIDS in San Francisco. Nature 1989; 338:251–253.
6. New York State Department of Health: AIDS in New York State. Albany, NY: New York State Department of Health, 1988.
7. Selwyn PA, Schoenbaum EE, Davenny K, *et al:* Prospective study of human immunodeficiency virus infection and pregnancy outcome in intravenous drug users. JAMA 1989; 261:9:1289–1294.
8. Johnstone FD, MacCallum L, Brettle R, Inglais JM, Peutherer JF: Does infection with HIV affect the outcome of pregnancy? Br Med J 1988; 296:467.
9. Robertson JR, Bucknall AB: Pregnancy and HTLVIII/LAV transmission in heroin users. Health Bull 1986; 44(6):364–366.
10. Smith PF, Mikl J, Truman BI, *et al:* HIV infection among women entering the New York State correctional system. Am J Public Health 1991; 81(Suppl):35–40, Ch VI.

VI. HIV Infection among Women Entering the New York State Correctional System

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Introduction

Human immunodeficiency virus (HIV) infection is becoming a leading medical problem among prison inmates in the United States, especially in states where the infection is prevalent. By October 1988 a total of 3,136 AIDS cases had been reported among inmates in a survey of 70 federal, state, and local correctional systems.[1] In New York State, which has more AIDS cases than any other state,[2] 1,046 cases had been reported among inmates of the state system by September 30, 1989.[3]

Because of the continuous turnover of prisoners, it is difficult to determine the precise number of persons with AIDS in New York State prisons at any time. However, a survey by the New York State Department of Correctional Services estimated that in September 1989 there were more than 800 inmates with symptomatic HIV disease and that approximately 550 inmates were on zidovudine therapy (Dr. Robert Greifinger, New York State Department of Correctional Services, personal communication). AIDS and HIV-related illness became the leading causes of death among inmates in 1985 and accounted for 68 percent of inmate deaths in 1987.[4]

In addition to severe HIV disease, there is evidence that many New York inmates are asymptomatically infected with HIV. A 1987 study of men entering the main intake center found that 17.4 percent were HIV-seropositive, the highest rate reported in any blind seroprevalence study of prisoners.[1,5] For intravenous drug users the rate was 44 percent. This study suggested that approximately 2,200 of the 12,600 men who entered this center during 1988 were infected with HIV.

HIV infection is also a problem among female prisoners in New York. Females are 4 percent of all inmates and have accounted for 4 percent of all inmate cases (41 cases). Thus the cumulative incidence rate from 1981 through 1988 has been equal for male and female inmates (27 cases per 1,000 inmates).[4]

Although the prevalence of HIV infection among female prisoners had never been studied, the statewide newborn HIV seroprevalence data suggested a major HIV problem among women in New York. This study was initiated to assess the extent of and risk factors for HIV infection among women entering the state prison system. Secondary objectives were to determine the association of HIV infection with clinical markers for syphilis, hepatitis B, and tuberculosis and to compare the fertility of seropositive and seronegative women.

Background

The New York State correctional system is the third largest in the United States, with 56 facilities housing 44,637 inmates at the end of 1988 (Mr. Gerald Bala, New York State Department of Correctional Services, personal communication). Of the approximately 1,800 female inmates, 69 percent come from New York City and 8 percent from the four counties closest to New York City. Forty-seven percent of female inmates are Black, 34 percent Hispanic, and 19 percent White. Nearly half (46 percent) have been convicted of drug-related offenses. The median duration of incarceration for women is 24 months (range, 12 to 900 months).

During 1988 a total of 1,017 new female inmates entered the New York State system. All female inmates enter at one central processing facility, where a medical evaluation is completed. The intake process includes a complete medical history, physical examination, and routine laboratory screening tests. Through 1988,

TABLE VI-1—Characteristics of Women Entering New York State Prisons, by Study Inclusion Status, September-December 1988

Characteristics	Included in Study (N=480)	Excluded from Study (N=40)	Difference (95% Confidence Interval)
Mean Age (Years)	29.4 (479)*	29.6 (40)*	+ 0.2 (− 2.6, 2.2)
Mean Education (Years)	10.2 (472)	10.4 (40)	+ 0.2 (− 0.8, 0.4)
Race/Ethnicity			
% Black	44.8 (215/480)	52.5 (21/40)	+ 7.7 (− 8.4, 23.8)
% Hispanic	37.5 (180/480)	32.5 (13/40)	− 5.0 (−20.1, 10.1)
% White	17.7 (85/480)	15.0 (6/40)	− 2.7 (−14.3, 8.9)
% With Residence in New York City	74.6 (344/461)	84.6 (33/39)	+10.0 (− 2.0, 22.0)
% Intravenous Drug Users	28.6 (136/475)	47.6 (10/21)	+19.0 (− 2.8, 40.7)

*For mean age and education, the numbers in parentheses indicate the number of women with available information. Fractions in the parentheses for the other variables indicate the number of women with each characteristic over the total number for whom information was available.

inmates were tested for HIV antibodies only when there was a medical indication, such as clinical evidence of immunodeficiency. In 1989 the testing policy was broadened to encourage all inmates, especially those with known risk factors, to undergo voluntary testing, regardless of their clinical status, so that appropriate counseling and medical therapy could be provided. The system does not segregate inmates on the basis of their HIV serologic status.

Methods

All women entering the New York State correctional system from September through December 1988 were included in this study. During this period no woman entered the system more than once, ensuring that there were no duplicates. Demographic information was abstracted from computerized prison records, and selected information that was routinely collected at prison intake regarding medical history, fertility, intravenous drug use, and other HIV risk factors was abstracted from each woman's prison medical record by trained personnel from the State Departments of Health and Correctional Services. After routine screening for syphilis, hepatitis B, and tuberculosis was performed and the results were abstracted to the data collection forms, information that identified specific inmates was removed from the forms, and a unique random number was used to link each form with the blood samples for HIV serologic testing. This process assured that the HIV test results could not be linked to specific inmates.

Syphilis screening was performed with the rapid plasma reagin test and, for confirmation of reactive specimens, the fluorescent treponemal antibody-absorption (FTA-ABS) test. Hepatitis B surface antigen (HBsAG) was determined by radioimmunoassay. These tests were performed by standard techniques at a commercial laboratory. Tuberculosis screening was performed with the intradermal tuberculin purified protein derivative Mantoux skin test (5 tuberculin units). Induration of 10 mm or greater was considered a positive reaction.

HIV testing was performed by the New York State Wadsworth Center for Laboratories and Research. Specimens were first tested in a single standard HIV-1 enzyme-linked immunosorbent assay (ELISA) (DuPont). Specimens with color development less than test cutoff values were recorded as ELISA-negative and were not tested further. ELISA-positive specimens were examined in a standard Western blot confirmatory assay (DuPont), and seropositivity was determined according to criteria recommended by the Association of State and Territorial Public Health Laboratory Directors and the Centers for Disease Control.[6]

The data from the forms and results of HIV serologic testing were merged and analyzed with computer software from SAS Institute, Inc.,[7] BMDP Statistical Software, Inc.,[8] and the Centers for Disease Control.[9] Chi-square, Fisher exact, and *t* tests were applied to univariate analysis. Standard methodology was used to calculate the 95 percent confidence intervals for differences in proportions[10] and differences in means.[11] Multivariate analysis to assess risk factors for HIV seropositivity utilized logistic regression. For this analysis women who reported having had sex with an intravenous drug user, bisexual male, or persons with AIDS or HIV infection or who had exchanged sex for money or drugs were classified as having practiced high-risk sex. Potential confounding of the estimated mean difference of live births and pregnancies between seropositive and seronegative women was investigated with a one-variable and two-variable stratified analysis.[12]

Results

During the four-month study period, 520 women entered the New York State prison system through its central processing facility. Of these, 480 women comprised the study group, while 25 were excluded because of missing demographic and medical information and 15 because of insufficient blood for HIV testing. Compared with the study group, a greater proportion of the 40 excluded women were Black, residents of New York City, and/or intravenous drug users (Table VI-1).

HIV Status and Risk Behaviors of the Study Group

In the study group 90 (18.8 percent) women were seropositive by ELISA; all were confirmed positive by Western blot. Maximum and minimum seroprevalence rates were estimated for all 520 women. Serologic results were available for 25 of the 40 excluded women: four were confirmed positive, one was positive by ELISA but indeterminant by Western blot, and 20 were negative. Thus the maximum estimated number of seropositive women was 110 (21.2 percent) of the 520 entrants: 90 in the study group plus four seropositives, one indeterminant, and 15 with insufficient blood for testing in the excluded group. If the 15 women with insufficient blood for testing and the woman with indeterminant results are assumed to be seronegative, the minimum estimated number of seropositive women was 94 (18.1 percent).

Of the 90 seropositive women in the study group, 53 were Hispanic, 31 Black, and six White. Eighty were between the ages of 20 and 39 years, and 82 reported New York City as their residence at the time of arrest. Sixty-one (67.8 percent) reported a history of intravenous drug use, and an additional six (6.7 percent) reported sexual contact with persons at risk for HIV infection. The remaining 23 reported no recognized risk behaviors for HIV infection, although six had a positive syphilis serology and another reported exchanging sex for money or drugs (suggesting prior high-risk sexual activity). An ad-

TABLE VI-2—HIV Seropositivity of Women Entering New York State Prisons by Demographic Characteristics, Potential Risk Factors, and Clinical Markers for Syphilis, Hepatitis B, and Tuberculosis, September-December, 1988.

Demographic, Risk Factor, and Clinical Marker Variables	Number Tested* (N=480)	Number HIV Positive (%) (N=90) (18.8%)	Odds Ratio** (95% Confidence Interval)
Demographic Characteristics			
Age (years)			
17–19	17	2 (11.8)	Referent
[illegible]–29	259	40 (15.4)	1.4 (0.3, 12.8)
30–39	160	40 (25.0)	2.5 (0.5, 23.4)
40–49	34	6 (17.6)	1.6 (0.2, 18.0)
50+	9	1 (11.1)	0.9 (0.0, 20.8)
Race/Ethnicity			
White, not Hispanic***	85	6 (7.1)	Referent
Black, not Hispanic	215	31 (14.4)	2.2 (0.9, 6.8)
Hispanic	180	53 (29.4)	5.5 (2.2, 16.3)
Education (years)			
≥13	14	1 (7.1)	Referent
10–12	313	52 (16.6)	2.6 (0.4, 112.2)
≤9	145	36 (24.8)	4.3 (0.6, 187.5)
Residence at Time of Arrest			
New York State, Excluding New York City	117	6 (5.1)	Referent
New York City	344	82 (23.8)	5.8 (2.4, 16.7)
Bronx	62	16 (25.8)	6.4 (2.2, 21.1)
Brooklyn	77	24 (31.2)	8.4 (3.1, 26.3)
Manhattan	120	32 (26.7)	6.7 (2.6, 20.4)
Queens	68	10 (14.7)	3.2 (1.0, 11.2)
Richmond	17	0 (0.0)	0 (0.0, 6.1)
Birthplace			
USA—Mainland	377	62 (16.4)	Referent
Caribbean	81	26 (32.1)	2.4 (1.3, 4.2)
South and Central America	15	2 (13.3)	0.8 (0.1, 3.6)
Potential Risk Factors			
Intravenous Drug Use			
No	339	27 (8.0)	Referent
Yes	136	61 (44.9)	9.4 (5.4, 16.4)
Sex with IVDU			
No	378	53 (14.0)	Referent
Yes	94	36 (38.3)	3.8 (2.2, 6.5)
Sex with Bisexual Male			
No	452	83 (18.4)	Referent
Yes	18	5 (27.8)	1.7 (0.5, 5.3)
Sex with Person with AIDS/HIV			
No	468	88 (18.8)	Referent
Yes	4	1 (25.0)	1.4 (0.0, 18.2)
Exchange of Sex for Money or Drugs			
No	426	75 (17.6)	Referent
Yes	47	13 (27.7)	1.8 (0.8, 3.7)
Blood Transfusion†			
No	469	85 (18.1)	Referent
Yes	9	5 (55.6)	5.7 (1.2, 28.9)
Evidence of Tattoo			
No	360	54 (15.0)	Referent
Yes	113	32 (28.3)	2.2 (1.3, 3.8)
Clinical Markers			
Syphilis Serology			
Negative	394	60 (15.2)	Referent
Positive	71	24 (33.8)	2.8 (1.6, 5.2)
Hepatitis B Surface Antigen			
Negative	420	78 (18.6)	Referent
Positive	10	3 (30.0)	1.9 (0.3, 8.5)
Tuberculin Skin Test			
Negative	401	77 (19.2)	Referent
Positive	65	13 (20.0)	1.1 (0.5, 2.1)

*Numbers do not always total to 480 due to missing information.

**Odds ratio for HIV seropositivity among women in each subgroup compared with the referent subgroup.

***Includes one Native American

†Since the dates of blood transfusions were not available, all women who had ever received a blood transfusion were considered to have this risk factor.

ditional 11 reported using street drugs nonintravenously, and two others had received tattoos. The remaining three did not report any of these behaviors nor have reactive syphilis serologies.

Associations of Risk Factors with HIV Seropositivity

HIV seroprevalence varied with demographic characteristics (Table VI-2). Women ages 30 to 39 years had the highest rate (25.0). Compared with the rate for Whites, seroprevalence was twice as high among Blacks and four times higher among Hispanics. Seroprevalence increased with decreasing levels of education. Women who resided in New York City at the time of arrest had higher rates than women from the rest of the state. Nearly one-third of women born in Caribbean countries were seropositive.

HIV seropositivity was associated with several potential risk factors for which information was available (Table VI-2). Of the 136 women who reported a history of intravenous drug use, 45 percent were HIV-seropositive, compared with 8 percent of the 339 women who denied intravenous drug use (odds ratio = 9.4). Seroprevalence was also strongly associated with a history of sex with an intravenous drug user, a history of blood transfusion, and evidence of a tattoo on physical examination.

Overall, of women with available test results, 15.3 percent had positive syphilis serology, 2.3 percent were positive for HBsAG, and 13.9 percent had positive tuberculin tests. Of these three clinical markers, a positive syphilis serology was most strongly associated with HIV seropositivity (Table VI-2).

For 472 women, information was available regarding their use or nonuse of specific street drugs (Table VI-3). Among the women denying any drug use, 7.5 percent were seropositive, compared with 8.2 percent of the women who used drugs nonintravenously and 45.9 percent of the women who admitted to intravenous drug use. Among intravenous drug users, those who used cocaine but not heroin had the lowest HIV seroprevalence (25.0 percent), users of heroin and not cocaine had an intermediate seroprevalence (43.6 percent), and users of both cocaine and heroin had the highest seroprevalence (51.3 percent). The corresponding percentages among drug users who denied intravenous use were lower but showed the same increasing trend from "cocaine, no heroin" to "cocaine and heroin."

TABLE VI-3—HIV Seropositivity of Women Entering New York State Prisons by History of Intravenous Drug Use and Type of Drug Used, September-December 1988.*

IV Drug Use Status and Type of Drug Used	Number Tested	Number HIV Positive (%)	Odds Ratio (95% Confidence Interval)	
Women Denying any Drug Use	107	8 (7.5)	Referent	
Women Using Drugs				
Non-Intravenously Only	232	19 (8.2)	1.1	(0.4, 2.9)
Cocaine, no Heroin	104	3 (2.9)	0.4	(0.1, 1.6)
Heroin, no Cocaine	30	5 (16.7)	2.5	(0.6, 9.4)
Cocaine and Heroin	19	4 (21.1)	3.3	(0.6, 14.1)
Drugs other than Cocaine and Heroin	79	7 (8.9)	1.2	(0.4, 3.9)
Women Using Drugs				
Intravenously	133	61 (45.9)	10.5	(4.5, 25.4)
Cocaine, no Heroin	16	4 (25.0)	4.1	(0.8, 18.2)
Heroin, no Cocaine	39	17 (43.6)	9.6	(3.4, 28.0)
Cocaine and Heroin	78	40 (51.3)	13.0	(5.3, 33.4)

*Eight women (three who admitted to intravenous drug use) were excluded because of incomplete or inconsistent information.

TABLE VI-4—Variables Associated with HIV Seropositivity by Logistic Regression Analysis* for Women Entering New York State Prisons, September-December 1988

Independent Variables	Adjusted Odds Ratio** (95% Confidence Interval)	
Intravenous Drug Use	9.4	(4.8, 18.3)
Race/Ethnicity		
Hispanic	5.8	(1.8, 18.0)
Black	3.3	(1.0, 10.6)
Positive Syphilis Serology	3.2	(1.6, 6.7)
New York City Residence	3.2	(1.1, 9.2)
Blood Transfusion***	2.3	(0.4, 14.5)
Evidence of Tattoo	1.6	(0.8, 3.1)
High Risk Sex†	1.3	(0.7, 2.6)

*The regression model included the seven variables shown, as well as age, as a continuous variable.

**The referent group for Hispanic and Black women was White women, and for women with New York City residence was women with residence in New York State excluding New York City.

***Since the dates of blood transfusions were not available, all women who had ever received a blood transfusion were considered to have this risk factor.

†Women who reported having had sex with an intravenous drug user, bisexual male, person with AIDS or HIV infection, or who exchanged sex for money or drugs were classified as having practiced high risk sex.

TABLE VI-5—Fertility of Women Entering New York State Prisons by HIV Serologic Status, September-December 1988

Measure of Fertility	HIV Positive Women	HIV Negative Women
Mean Number of Pregnancies/Woman* (range)	2.7 (0–8)	2.7 (0–13)
Mean Number of Live Births/Woman (range)	1.8 (0–6)	1.7 (0–8)
Fraction of Women Nulliparous (%)	20/90 (22.2)	113/390 (30.3)

*Two seropositive and 15 seronegative women with incomplete pregnancy histories were excluded from this analysis.

Multivariate analysis identified five factors associated with HIV seropositivity (Table VI-4): women who were intravenous drug users, of Hispanic or Black race/ethnicity, with a positive syphilis serology, or residents of New York City were more likely to be seropositive than women without these factors. There were no interaction effects among any of the variables.

Fertility

Of 21 women who were known to be pregnant, two (9.5 percent) were HIV positive. The mean number of pregnancies, the mean number of live births, and the percentage of women who were nulliparous were similar for HIV-positive and HIV-negative women (Table VI-5). Stratified analysis to control for differences in age, racial composition, and intravenous drug use between seropositive and seronegative women did not change these findings (data not shown).

Discussion

This study, as well as an earlier study of male prison entrants,[5] was crucial in defining the magnitude of the HIV epidemic among New York State prisoners, describing its epidemiology, and promoting increased public health attention to address the epidemic in this high-risk population.

HIV seroprevalence was higher among male (17.4 percent) and female (18.8 percent) entrants to New York State prisons than has been previously reported in any mass screening program or blind epidemiologic study among prisoners in the United States.[1] The seroprevalence among all 520 female entrants during the study period may have actually been closer to our maximum estimate of 21 percent, because many of the excluded women with insufficient blood samples for HIV testing may have had scarred veins from intravenous drug use and been at increased risk for HIV infection. Most seroprevalence rates reported among prisoners in other jurisdictions have been less than 3 percent, with the notable exception of high rates for males (7.0 percent) and females (15.4 percent) entering Maryland State prisons in 1985.[1] The Maryland study found that seroprevalence among male entrants was constant from 1985 to 1987.[13] Follow-up studies of New York State inmates are planned to determine trends in seroprevalence with time.

The blinded design of our study precluded collection of extensive risk information, and the cross-sectional design prevented assessment of temporal associations between HIV infection and risk factors or other infectious diseases. However, the high rate of HIV seropositivity among female inmates is consistent with their relatively high frequency of known risk behaviors, especially intravenous drug use, and the fact that most of them come from New York City, where HIV infection is prevalent. In addition to intravenous drug use, we found that a positive syphilis serology was strongly associated with HIV positivity. Of the 23 HIV-positive women who denied any known risk behavior, six had a positive syphilis test. The association between genital ulcer disease and HIV infection has been previously reported.[14–16] Genital ulcer disease may be a marker for prior high-risk sexual activity and may increase the risk of HIV transmission by disrupting the vaginal or cervical mucosa.

Although HIV infection has not been shown to be associated with tattooing,[17,18] HIV transmission through tattoo needles is biologically plausible, and the possible spread of HIV by this route to prisoners has been reported.[19] In our study, women with tattoos were twice as likely to be HIV-seropositive as women without tattoos (Table VI-2). However, when other possible exposures were controlled in the multivariate analysis, tattoos were not associated with HIV positivity, suggesting that tattooing was not a major factor for transmission in this population.

Our study showed an association between high HIV seroprevalence and Black or Hispanic ethnicity. This association has been described previously among intravenous drug users and attributed to differences in the social setting of drug use and specific drug use behaviors for persons of different ethnic backgrounds.[20–22] Our results may have been affected by these factors, as well as by other unmeasured risk behaviors, such as sexual contact with a person not recognized as being at high risk for HIV infection.

Although other studies have found that intravenous cocaine use is more strongly associated with HIV infection than heroin use,[21,23] our results suggested the opposite (Table VI-3). However, the small number of women in our study limited its power to detect differences by drug use. In any case, caution is needed in interpreting these results, since we had no information on the frequency, quantity, or pattern of intravenous drug use, or on the frequency of high-risk sexual activity.

HIV-positive women in our study reported the same number of pregnancies and live births as the seronegative women, and similar percentages of each group were nulliparous (Table VI-5), even after controlling for differences in age, race, and intravenous drug use between the two groups. These findings suggest that HIV infection did not affect fertility, but because we had no information on the timing of HIV seroconversion relative to the reported pregnancies, the exact relationship between HIV infection and fertility was impossible to assess. Our results are consistent with those of a study among New York City intravenous drug users which found that asymptomatic HIV infection was not associated with a decreased pregnancy rate or an increased risk of adverse pregnancy outcomes.[24] Another study of 136 pregnancies among British women also found no evidence that HIV infection affected the outcome of pregnancy, although there were more spontaneous abortions among the seropositive women.[25]

Determining the impact of HIV infection on fertility would provide important information for interpreting the blind HIV newborn seroprevalence studies that are being conducted widely. These studies provide nearly complete testing of all newborns and therefore are useful measures of HIV-infection among women giving birth and, by extrapolation, among all women.[26] However, if HIV infection substantially reduces fertility, infected women would be less likely to deliver babies, and newborn seroprevalence studies would underestimate infection rates among women. Current evidence suggests that asymptomatic HIV infection may not reduce fertility, but additional studies are needed.

After our study, funding was increased to enable four teams of counselors to provide prisoner education regarding HIV infection and to encourage all prisoners to consider HIV antibody testing. Our study and the earlier study of male inmates[5] also helped to quantify the need for increased medical services for HIV-infected prisoners. Applying the seroprevalence rates from these two studies to the number of entrants at the two prison centers, we estimate that in 1988 approximately 2,200 men and 200 women entered New York State prisons with HIV infection. In addition to routine medical care, these inmates need medical evaluation for newly approved therapies for asymptomatic HIV infection and adequate follow-up of other associated medical conditions, especially tuberculosis, which has shown an alarming increase in association with HIV infection.[27] The problem of perinatal transmission in prison was also apparent from our study, since two of the 21 pregnant women were HIV positive. To address these needs, provision for increased medical care is being planned in New York State prisons; additional seroprevalence studies will be conducted to monitor the HIV epidemic among prisoners in the future.

Summary

Human immunodeficiency virus infection is the leading medical problem among prison inmates in several states. In 1988 a blinded seroprevalence study was conducted on 480 New York female prison entrants to determine the prevalence of and risk factors for HIV infection in this population. Ninety (18.8 percent) women were HIV-seropositive. Seroprevalence was highest among women ages 30–39 (25.0 percent) and varied by ethnicity (Hispanics, 29.4 percent; Blacks, 14.4 percent; Whites, 7.1 percent) and residence (New York City, 23.8 percent; Upstate, 5.1 percent). Nearly half (44.9 percent) of the 136 acknowledged intravenous drug users and one-third (33.8 percent) of the 71 women with a positive syphilis serology were HIV-seropositive. There was no difference in fertility histories between seropositive and seronegative women, and two of 21 pregnant women were seropositive. This study led to increased clinical and prevention services for this high-risk population. ☐

Acknowledgments

The authors gratefully acknowledge the staff of the Bedford Hills Correctional Facility and Esta Blum of the New York State Department of Health for assisting in data collection.

References

1. Hammett TM: Issues and Practices. 1988 Update: AIDS in Correctional Facilities. Washington, DC: Abt Associates Inc, 1989.
2. Centers for Disease Control; HIV/AIDS Surveillance Report. Atlanta, GA: CDC, October 1989.
3. New York State Department of Health: AIDS Surveillance Monthly Update. Albany, NY: New York State Department of Health, September 1989.
4. Morse DL, Truman B, Mikl J, Smith P, Broaddus R, Maguire B: The epidemiology of AIDS among New York State prison inmates. Abstract ThDP45, presented at the V International Conference on AIDS, Montreal, Canada, June 1989.
5. Truman BI, Morse D, Mikl J, Lehman S,

Forte A, Broaddus R, Stevens R: HIV seroprevalence and risk factors among prison inmates entering New York State prisons. Abstract 4207, presented at the IV International Conference on AIDS, Stockholm, Sweden, June 1988.

6. Centers for Disease Control: Interpretation and use of the Western blot assay for serodiagnosis of human immunodeficiency virus type 1 infections. MMWR 1989; 38 (suppl no. S-7):1–7.
7. SAS Institute Inc: SAS User's Guide: Basics: Version 5 Edition. Cary, NC: SAS Institute Inc, 1985.
8. Dixon WJ (ed), Brown MB, Engelman L, Hill MA, Jennrich RI: BMDP Statistical Software Manual. Berkeley: University of California Press, 1988.
9. Dean JA, Dean AG, Burton A, Dicker R: Epi Info, Version 3. Atlanta, GA: Centers for Disease Control, January 1988.
10. Fleiss JL: Statistical Methods for Rates and Proportions. New York: John Wiley and Sons, 1981.
11. Dixon WJ, Massey FJ: Introduction to Statistical Analysis. New York: McGraw-Hill, 1969.
12. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research. Belmont, CA: Wadsworth, 1982.
13. Vlahov D, Brewer F, Munoz A, Hall D, Taylor E, Polk BF: Temporal trends of human immunodeficiency virus type 1 (HIV-1) infection among inmates entering a statewide prison system, 1985–1987. J AIDS 1989; 2:283–290.
14. Kreiss JK, Koech D, Plummer FA, *et al:* AIDS virus infection in Nairobi prostitutes. Spread of the epidemic to East Africa. N Engl J Med 1986; 314:414–418.
15. Greenblatt RM, Lukehart SA, Plummer FA, *et al:* Genital ulceration as a risk factor for human immunodeficiency virus infection. AIDS 1988; 2:47–50.
16. Stamm WE, Handsfield HH, Rompalo AM, Ashley RL, Roberts PL, Corey L: The association between genital ulcer disease and acquisition of HIV infection in homosexual men. JAMA 1988; 260:1429–1433.
17. Castro KG, Lifson AR, White CR, *et al:* Investigations of AIDS patients with no previously identified risk factors. JAMA 1988; 259:1338–1342.
18. The Collaborative Study Group of AIDS in Haitian-Americans: Risk factors for AIDS among Haitians residing in the United States. Evidence of heterosexual transmission. JAMA 1987; 257:635–639.
19. Doll DC: Tattooing in prison and HIV infection. (letter) Lancet 1988; 1:66–67.
20. Schoenbaum EE, Hartel D, Selwyn PA, *et al:* Risk factors for human immunodeficiency virus infection in intravenous drug users. N Engl J Med 1989; 321:874–879.
21. Chaisson RE, Bacchetti P, Osmond D, Brodie B, Sande MA, Moss AR: Cocaine use and HIV infection in intravenous drug users in San Francisco. JAMA 1989; 261:561–565.
22. Des Jarlais DC, Friedman SR: HIV infection among persons who inject illicit drugs: problems and prospects. J AIDS 1988; 1:267–273.
23. Friedman SR, Rosenblum A, Goldsmith D, *et al:* Risk factors for HIV-1 infection among street-recruited intravenous drug users in New York City. Abstract TA012, presented at the V International Conference on AIDS, Montreal, Canada, June 1989.
24. Selwyn PA, Schoenbaum EE, Davenny K, *et al:* Prospective study of human immunodeficiency virus infection and pregnancy outcomes in intravenous drug users. JAMA 1989; 261:1289–1294.
25. Johnstone FD, MacCallum L, Brettle R, Inglis JM, Peutherer JF: Does infection with HIV affect the outcome of pregnancy? Br Med J 1988; 296:467.
26. Lessner L: Projection of AIDS incidence in women in New York State. Am J Public Health 1991; 81(Suppl):30–34 Ch V.
27. Braun MM, Truman BI, Maguire B, *et al:* Increasing incidence of tuberculosis in a prison inmate population. JAMA 1989; 261:393–397.

VII. HIV Seroprevalence in Clients of Sentinel Family Planning Clinics

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Introduction

Family planning clinics serve predominantly young, sexually active clients, a substantial proportion of whom may be infected—or at risk of infection—with human immunodeficiency virus (HIV). The purposes of this study were to document the prevalence of HIV antibody in women attending sentinel family planning clinics and to monitor the trends in infection over time.

In New York State, there are approximately 200 discrete family planning clinics, located in all counties except Putnam (where negotiations for a clinic are in progress). All but one of these clinics are publicly subsidized. Other groups and/or individuals provide family planning services but d[illegible] have a clinic dedicated solely for t[illegible]pose.

Fam[illegible] planning clinics provide medically approved methods of contraception, medical evaluations, screening and treatment for vaginal infections and sexually transmitted diseases, level-I screening for infertility, and voluntary HIV counseling and testing services.

Most clients in New York State are women. Approximately 75 percent are under age 25. Overall 21 percent are Black and 13 percent Hispanic. [In New York State approximately 14 percent of the population is Black and 12 percent of Hispanic origin (Current Population Survey: March 1988)].

One constraint on this study was that relatively few of the clinics routinely obtain blood specimens. Random sampling of clients and/or clinics was therefore not feasible. Instead the qualifying clinics were considered sentinels, and the results were adjusted for extrapolation to the total population of clients.

Methods

The study was conducted over a period of 26 months, from February 1, 1988 through March 31, 1990.

The study sites were limited to publicly subsidized family planning clinics that routinely drew whole blood specimens for syphilis serology from clients during the initial medical evaluation. Clinics throughout the state were screened for this practice. In early 1988, 38 clinics met this criterion, and 37 agreed to participate. Of the participating facilities, 13 were located in New York City boroughs and the rest in other counties: Westchester, seven; Nassau, four; Onondaga, three; Monroe, five; Orleans, one; Genesee, one; and Erie, three. One clinic in Monroe County was closed shortly after the study was initiated. Five additional clinics (Rockland, two; Orange, two; Nassau, one) entered the study after changes in their routine blood-collection practices.

Study subjects were limited to clients undergoing an initial medical evaluation for contraceptive services. Clients seen for annual or follow-up visits were excluded to avoid repeat testing of the same individuals over time. Individuals seen solely for pregnancy testing were also excluded, because blood specimens were not routinely obtained on this population. Clients seen only for HIV counseling and testing were excluded to minimize self-selection bias.

If a client met these criteria, a seroprevalence study test requisition (SSTR) form was completed by the clinic staff and submitted along with the routine laboratory request form and blood specimen to a contract laboratory. The completed form contained the client's age, race or ethnicity, and county and zip code of residence, as well as the clinic site and the month and year of specimen collection. Identifying information (e.g., name, street address, or medical record number) was not collected; thus the information could not be traced back to the individual client.

If serum was left over after all routine laboratory tests were performed, the contract laboratory would pipette at least 0.5 ml into a tube labeled with the blind seroprevalence number and send the serum with the SSTR form to the New York State Department of Health's Wadsworth Center for Laboratories and Research. No additional blood was drawn for this study.

When the blood drawn was insufficient for blind HIV testing, the SSTR alone was sent to the Department of Health. These data were used to estimate any bias from the loss of specimens. The data were not used for any other analysis.

The Wadsworth Center performed an ELISA (enzyme-linked immunosorbent assay) (DuPont) for HIV antibody. ELISA reactives were confirmed with a Western blot (in-house assay until May 1988, then DuPont). All Western blots were interpreted by the latest Association of State and Territorial Public Health Laboratory Directors criterion[1] for reactive specimens, i.e. virus-specific bands in at least two of the following major band locations: gp160/120, gp41, or p24. Specimens with other major virus-specific bands were interpreted as equivocal. All other specimens were considered nonreactive.

Seroprevalence rates were calculated as: (number of Western blot reactives)/(total number of specimens minus equivocals). Because the specimens could not be traced to individual clients, we could not request second samples for reevaluation of equivocals.

Age-specific seroprevalence rates were calculated in five-year intervals for ages 15 to 39. Individuals under age 15 were grouped, as were those over age 39. Racial/ethnic-specific seroprevalence rates were calculated for non-Hispanic Whites, non-Hispanic Blacks, Hispanics, Others, and Unknowns. Others and Unknowns had to be combined due to small cell sizes for some combinations of age and race/ethnicity groups.

HIV seroprevalence rates by quarter-year of clinic visit were calculated to describe changes in seroprevalence over time. For this analysis three of the 37 initial clinics were eliminated: one that closed early in the study and two that closed later. Trends were calculated only for the 34 clinics that participated throughout the entire study period.

To estimate the completeness of the data set, we used the number of initial medical examinations in 1988 reported by each participating clinic to the New York State Department of Health's Bureau of Reproductive Health. This number was compared to the number of specimens submitted during the first year of the study (February 1988–January 1989). Clinic records were available from all but one site in upstate New York.

Bureau of Reproductive Health statistics were also used to adjust for differences between the sentinel clinic clients and the overall family planning clinic population. The age, racial/ethnic, and residence distributions of the two groups were compared and the HIV seroprevalence rates were adjusted directly for differences, using the overall population as the standard.

The results of the sentinel clinic study were compared to the results of the Newborn HIV Seroprevalence Study. Sentinel clinic rates were adjusted for the differences in age, racial/ethnic, and residence distributions. The adjusted rates were calculated for New York City, New York State exclusive of New York City, and statewide, using the distribution of childbearing women as the standard population.

Between February and July 1988 a separate, unlinked HIV risk assessment questionnaire was administered to clients of 30 family planning clinics by clinic staff. Participation in the risk study was voluntary and not limited to the subset of women seen for an initial medical evaluation. The results were submitted to the New York State Department of Health without patient identifiers. Participation was voluntary on the part of the facility and the clients.

The questionnaire included items on the client's age; race/ethnicity; county of residence; history of intravenous drug use; history of living in a high-risk country (as defined by the Centers for Disease Control); history of being a sex partner of a bisexual male, drug user, hemophiliac, or person with AIDS or HIV infection; and maximum number of sex partners in any six-month period since 1978 (0, 1, 2, 3, or 4+).

Results

Sentinel Clinic Results

Between February 1, 1988 and March 31, 1990, a total of 28,745 specimens were obtained from women having initial medical examinations in the sentinel family planning clinics. Of these specimens, 896 (3 percent) had an insufficient quantity of serum left over for analysis. An additional 1 percent were not tested due to improper shipping, leakage of tubes, or blank forms or because the specimen was obtained from a male client. Twelve (0.04 percent) had equivocal HIV test results and were eliminated from the analysis. Of the 27,549 remaining specimens 144 (0.52 percent) were HIV antibody-positive.

Fourteen of the 41 clinic sites (one in New York City, 13 in New York State exclusive of New York City), which contributed 11.8 percent of the study population, had no seropositives identified during the study period. Eight clinics (two in New York City) had rates of seropositivity between 0.01 and 0.50 percent; 11 clinics (six in New York City) had rates between 0.51 and 1.00 percent; and eight clinics (four in New York City) had rates in excess of 1 percent. The highest seroprevalence rate was 3.91 percent, detected in a mobile clinic in New York City.

The highest seroprevalence rate by residence of the client was detected among residents of New York City, with 0.82 percent identified as positive. The rates by boroughs (and the numbers tested/received) were: Manhattan, 0.73 percent (15/2,050); Bronx, 0.98 percent (20/2,031); Kings, 0.81 percent (50/6,187); Queens, 0.84 percent (20/2,387); and Staten Island, 0.67 percent (6/891).

Residents living elsewhere in the state had a 0.24 percent rate of seropositivity. Clients from counties nearest New York City (Westchester, Nassau, and Rockland) had five times the seroprevalence of residents from other counties (Erie, Monroe, Orleans, Genesee, Onondaga, and Orange): 0.54 versus 0.11 percent, respectively.

No seropositives were detected among the 805 individuals tested under the age of 15, and three seropositives (0.42 percent) were detected among the 722 individuals age 40 or more (Table VII-1). Between these groups HIV seroprevalence increased with age. Women 35–39 years old had approximately nine times the HIV seroprevalence rate of 15–19 year olds (1.56 versus 0.18 percent). The trend of increasing HIV seroprevalence with age was seen for all racial/ethnic groups.

Minority women (non-Hispanic Black or Hispanic) had an HIV seroprevalence of 0.76 percent (Table VII-1). This was about six times the rate detected among non-Hispanic Whites (0.13 percent).

HIV seroprevalence rates by quarter-year of clinic visit are shown in Table VII-2. The highest rates (0.63 and 0.71

TABLE VII-1—HIV Seroprevalence by Age and Racial/Ethnic Status of Sentinel Family Planning Clinic Clients, February 1988–March 1990

Age (years)	Percent (Number positive/number tested)				
	Non-Hispanic White	Non-Hispanic Black	Hispanic	Other/ Unknown	Total
A. New York City					
<15	0.00 (0/12)	0.00 (0/256)	0.00 (0/123)	0.00 (0/4)	0.00 (0/395)
15–19	0.00 (0/492)	0.25 (9/3,638)	0.30 (6/1,997)	0.00 (0/136)	0.24 (15/6,263)
20–24	0.89 (3/337)	1.07 (19/1,769)	0.88 (11/1,253)	0.00 (0/87)	0.96 (33/3,446)
25–29	1.26 (2/159)	1.92 (14/730)	2.09 (13/622)	1.56 (1/64)	1.90 (30/1,575)
30–34	1.28 (1/78)	2.17 (8/369)	2.26 (9/399)	3.23 (1/31)	2.17 (19/877)
35–39	3.45 (1/29)	1.83 (3/164)	2.05 (5/244)	11.11 (1/9)	2.24 (10/446)
>39	4.76 (1/21)	1.12 (1/89)	0.00 (0/317)	0.00 (0/8)	0.46 (2/435)
Unknown	0.00 (0/3)	0.00 (0/60)	7.41 (2/27)	0.00 (0/19)	1.83 (2/109)
Total	0.71 (8/1,131)	0.76 (54/7,075)	0.92 (46/4,982)	0.84 (3/358)	0.82 (111/13,546)
B. New York State Exclusive of New York City					
<15	0.00 (0/191)	0.00 (0/177)	0.00 (0/27)	0.00 (0/15)	0.00 (0/410)
15–19	0.03 (1/3,816)	0.42 (4/953)	0.27 (1/367)	0.00 (0/204)	0.11 (6/5,340)
20–24	0.04 (1/2,799)	0.46 (4/872)	0.50 (3/606)	0.00 (0/142)	0.18 (8/4,419)
25–29	0.10 (1/1,013)	1.31 (6/459)	0.49 (2/406)	0.00 (0/72)	0.46 (9/1,950)
30–34	0.41 (2/490)	1.93 (4/207)	0.00 (0/237)	0.00 (0/40)	0.62 (6/974)
35–39	0.00 (0/173)	2.47 (2/81)	0.88 (1/114)	0.00 (0/17)	0.78 (3/385)
>39	0.00 (0/131)	1.61 (1/62)	0.00 (0/86)	0.00 (0/8)	0.35 (1/287)
Unknown	0.00 (0/93)	0.00 (0/25)	0.00 (0/37)	0.00 (0/71)	0.00 (0/226)
Total	0.06 (5/8,706)	0.74 (21/2,836)	0.37 (7/1,880)	0.00 (0/569)	0.24 (33/13,991)
*C. Statewide**					
<15	0.00 (0/203)	0.00 (0/433)	0.00 (0/150)	0.00 (0/19)	0.00 (0/805)
15–19	0.02 (1/4,314)	0.28 (13/4,592)	0.30 (7/2,364)	0.00 (0/340)	0.18 (21/11,610)
20–24	0.13 (4/3,138)	0.87 (23/2,641)	0.75 (14/1,859)	0.00 (0/229)	0.52 (41/7,867)
25–29	0.26 (3/1,172)	1.68 (20/1,190)	1.46 (15/1,028)	0.74 (1/136)	1.11 (39/3,526)
30–34	0.53 (3/568)	2.08 (12/577)	1.41 (9/637)	1.41 (1/71)	1.35 (25/1,853)
35–39	0.50 (1/202)	2.04 (5/245)	1.68 (6/358)	3.85 (1/26)	1.56 (13/831)
>39	0.66 (1/152)	1.32 (2/151)	0.00 (0/403)	0.00 (0/16)	0.42 (3/722)
Unknown	0.00 (0/96)	0.00 (0/85)	3.12 (2/64)	0.00 (0/90)	0.60 (2/335)
Total	0.13 (13/9,845)	0.76 (75/9,914)	0.77 (53/6,863)	0.32 (3/927)	0.52 (144/27,549)

*Includes 12 clients for whom no county of residence was identified.

TABLE VII-2—HIV Seroprevalence by Quarter-Year of Clinic Visit for Sentinel Family Planning Clients, February 1988–March 1990

Quarter	Number Positive	Number Tested	Percent Positive
1st 1988	8	2,330	0.34
2nd 1988	19	3,033	0.63
3rd 1988	13	3,162	0.41
4th 1988	18	3,283	0.55
1st 1989	16	3,350	0.48
2nd 1989	13	3,010	0.43
3rd 1989	13	2,710	0.48
4th 1989	13	2,394	0.54
1st 1990	18	2,521	0.71

*Data limited to 34 clinics that participated during the entire study period.

percent) occurred in the second quarter of 1988 and first quarter of 1990. The lowest rate (0.34 percent) was detected in the incomplete first quarter of 1988. No trend over time is evident.

Extrapolation to All Family Planning Clinics

According to Bureau of Reproductive Health statistics, a statewide total of 68,644 initial medical examinations at family planning clinics were done in 1988. Of these examinations, 15,112 (22 percent) involved clients at the sentinel clinics. During the first year of the study (beginning in February 1988) 12,575 client specimens were included in the analysis. This is an estimated sampling rate of 83 percent in the sentinel clinics for 1988: 86 percent in New York City, 80 percent in New York State exclusive of New York City.

Sentinel clinic clients were more likely to be non-Hispanic Black (36.5 percent) or Hispanic (25.3 percent) than were clients in general (21.2 percent and 13.5 percent, respectively). The age distributions were similar: women under age 25 were 72.3 percent of clients at sentinel clinics and 73.4 percent at all clinics statewide.

After adjustments for differences in the racial/ethnic and residence distributions, the extrapolated HIV seroprevalence rates for all family planning clinic clients were: New York City, 0.82 percent; New York State exclusive of New York City, 0.15 percent; and statewide, 0.31 percent.

TABLE VII-3—Risk Factors for HIV Infection of Clients at Sentinel Family Planning Clinics, February 1988–July 1988

Risk Factor	Number Admitting	Number Responding	Percent Admitting
New York City			
IV drug use	13	4,692	0.28
SP* IVDU	47	4,691	1.00
SP* bisexual	35	4,703	0.74
SP* hemophiliac	4	4,680	0.09
High-risk country	28	838	3.34
SP* HIV+	24	4,642	0.52
Having ≥4 SP**	184	4,669	3.94
Any risks above	286	4,703	6.08
New York State Exclusive of New York City			
IV drug use	28	2,174	1.29
SP* IVDU	81	2,179	3.72
SP* bisexual	34	2,189	1.55
SP* hemophiliac	1	2,179	0.05
High-risk country	32	2,176	1.47
SP* HIV+	7	2,173	0.32
Having ≥4 SP**	194	2,141	9.06
Any risks above	297	2,189	13.57
Total			
IV drug use	41	6,866	0.60
SP* IVDU	128	6,870	1.86
SP* bisexual	69	6,892	1.00
SP* hemophiliac	5	6,859	0.07
High risk country	60	3,014	1.99
SP* HIV+	31	6,815	0.45
Having ≥4 SP**	378	6,810	5.55
Any risks above	583	6,892	8.46

*SP—Sexual partner(s).
**SP—Sexual partners in a six-month period since 1978.

Comparison to Newborn Seroprevalence Data

Because there were substantial differences between the demographic profiles of family planning clinic clients and those of childbearing women, adjusted seroprevalence rates were calculated using the age, racial/ethnic, and residence distributions of childbearing women as the standard.

The adjusted seroprevalence rates were: New York City, 1.53 percent; New York State exclusive of New York City, 0.25 percent; and statewide, 0.92 percent. These figures were 24 to 47 percent higher than the rates for childbearing women in the Newborn HIV Seroprevalence Study: New York City, 1.24 percent; New York State exclusive of New York City, 0.17 percent; and statewide, 0.66 percent.[2]

Risk Factors

Thirty sentinel clinics participated in the voluntary risk assessment study. Risk assessment forms were completed on 6,892 clients, who were generally of the same age (71 percent under age 25) as the whole study population. The risk-assessment group was more likely to reside in New York City and to be non-Hispanic Black (50 percent) or Hispanic (27 percent) than the overall sentinel population. (Of the risk-assessment group 21 percent were non-Hispanic White, and 3 percent were of Other or Unknown race/ethnicity.)

The number and percent of clients in the sentinel clinics admitting to the selected risk factors for HIV infection are presented in Table VII-3. The most frequently reported risk factors were having sex with four or more sex partners in a six-month period, having lived in a high-risk country, having had sex with an intravenous drug user, and having had sex with a bisexual male. Of all the participating clients, 583 (8.46 percent) admitted to at least one of the risk factors. Fifty-nine (11.64 percent) of the pregnant women admitted to at least one risk factor.

The women who volunteered for this study were not limited to those in the seroprevalence study. Not all sentinel clinics participated, and the extent of participation varied considerably among the participating clinics. For these reasons the results should be considered background information and not interpreted to explain seroprevalence results obtained for the full study population.

Discussion

Family planning clinics serve predominantly young, sexually active clients. In this study of 41 sentinel clinics, approximately one in 200 clients was infected with HIV.

The clinics in this study were not representative of all of the approximately 200 clinics in the state. Sentinel clinics were limited to those that routinely drew blood for syphilis serology on all initial clients. These clinics tended to be in areas with higher syphilis incidence rates and thus potentially higher HIV infection rates. The crude and adjusted HIV seroprevalence rates obtained in this study may overestimate the actual rate for the total family planning clinic population. The true rates are likely to be closer to those found in the Newborn HIV Seroprevalence Study.

A slightly low bias in statewide rates may have been introduced by the 896 specimens that were insufficient for testing. These specimens represented clients who resided primarily in New York City (79.7 percent). The 896 clients resembled the entire study group in their age distribution (71.5 percent under age 25) and resembled other New York City clients in their racial/ethnic distribution (non-Hispanic White, 21.0 percent; non-Hispanic Black, 42.5 percent; Hispanic, 34.4 percent; Other, 2.1 percent).

After reviewing the results of this and other seroprevalence studies involving New York State residents, the New York State Department of Health required HIV education, counseling, and testing programs as a condition of funding in all publicly subsidized clinics for family planning and prenatal care.

Since virtually all women seen in these clinics are sexually active, they are potentially at risk for HIV infection. Knowledge of an individual's HIV infection status is important to prevent transmission, particularly vertical transmission from mother to infant, and to prevent or defer certain complications of HIV infection with early therapeutic intervention.

In addition to providing HIV counseling and testing, family planning and prenatal care clinics must be prepared to provide or offer referral for care for their HIV-positive clients.

Summary

In February 1988 the New York State Department of Health initiated a study to determine the prevalence of HIV antibody in women attending selected, publicly subsidized family planning clinics. During a 26-month study period, 27,549 blood specimens were obtained from women having an initial medical examination in 41 clinic sites throughout the state. Of these clients 144 (0.52 percent) were seropositive.

The HIV seroprevalence rate increased with age to a high of 1.56 percent for 831 women ages 35 to 39. The seroprevalence rate for non-Hispanic Black or Hispanic clients (0.76 percent) was about six times the rate for non-Hispanic Whites (0.13 percent). No overall increasing or decreasing trend in prevalence of HIV infection was detected during the study period. ☐

Acknowledgments

The authors gratefully acknowledge the contribution of the clinical and laboratory staff of the participating clinics and laboratories.

References

1. Centers for Disease Control: Interpretation and use of the Western blot assay for serodiagnosis of Human Immunodeficiency Virus type 1 infections. MMWR 1989; 38(suppl S-7):1–7.
2. Novick LF, Glebatis DM, Stricof RL, MacCubbin PA, Lessner L, Berns DS: Newborn seroprevalence study: Methods and results. Am J Public Health 1991; 81(suppl):15–21, Ch II.

VIII. Hospitalization of HIV-Seropositive Newborns with AIDS-Related Disease within the First Year of Life

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Introduction

HIV seropositivity in a newborn is a reflection of the mother's HIV infection status, not necessarily that of the infant.[1,2] Only a minority of seropositive infants are actually infected, remaining HIV-positive after 18 months. Studies of perinatally acquired infection have shown wide variation in the rates of maternal transmission.[3-10] Factors such as low numbers of study subjects, loss to follow-up, neonatal deaths from indeterminate causes, absence of detectable antibodies in some infants with HIV-related illness,[10-12] and asymptomatic HIV-positive infants[9] erode investigators' ability to determine the true rate of vertical transmission of HIV infection.

This study was initiated in an attempt to measure the impact of HIV-seropositive newborns on the hospital care system. Our strategy was to determine the proportion of HIV-seropositive newborns who are hospitalized for an AIDS-related diagnosis (ARD) during their first year of life and to characterize those hospitalizations. This information can be used to plan the inpatient services required for HIV-infected infants.

Methods

The Statewide Planning and Research Cooperative System (SPARCS), maintained by the New York State Department of Health, collects diagnostic, demographic, and financial data on every hospital discharge in New York State.[13] Data on each hospital discharge include patient demographic information and primary and secondary diagnostic and procedure codes.

All hospital discharges from December 1987 through June 1989 were extracted from the SPARCS data base for any infant with a date of birth from December 1987 through June 1988 and an ARD of ICD-9CM 042, 043, 044, 795.8, or other selected codes (see Appendix) in any of the five diagnostic code categories. If a hospitalization had more than two ARDs, the code with the highest designation in the hierarchy listed above was used in the analysis.

Since SPARCS records represent hospital discharges and not individuals, multiple hospital discharges of an individual were collapsed into a single longitudinal case by linkages based on the date of birth, gender, and county of residence. Additional data elements such as dates of admission and discharge, medical record number, zip code of residence, race/ethnicity, patient disposition, and transfer status were examined to validate the case formation.

The number of HIV-seropositive infants who were born in each month from December 1987 to June 1988 was determined from the Newborn HIV Seroprevalence Study.[14]

The newborn seroprevalence and SPARCS data bases do not contain personal identifiers and cannot be linked. The analysis was therefore restricted to comparing, for each month of birth, the number of HIV-seropositive newborns and the number of infants hospitalized with an ARD within the first year of life. Frequencies were tabulated on the number of hospitalizations, the length of stay, each AIDS-related ICD-9-CM diagnostic code, age at first hospitalization, and case fatality rates.

Results

A total of 995 HIV-seropositive newborns born December 1987 through June 1988 were confirmed in the Newborn

TABLE VIII-1—Ratio of Infants Hospitalized for ARD within 12 Months after Birth to HIV-Seropositive Newborns, by Month of Birth

Month of Birth	ARD Cases	Number of HIV+ Newborns	Percent of ARD Cases/HIV+ Newborns
December 1987	26	169	15
January 1988	19	139	14
February 1988	18	122	15
March 1988	19	132	14
April 1988	24	135	18
May 1988	22	136	16
June 1988	23	162	14
Total	151	995	15

TABLE VIII-2—Hospitalizations within 12 Months of Birth by ARD at First Hospitalization

	Number of Hospitalizations		
	One	Two or More	
ARD at First Hospitalization	No. (Percent)	No. (Percent)	Total N
042	35 (73)	13 (27)	48
043/044	22 (79)	6 (21)	28
795.8	28 (64)	16 (36)	44
Other	25 (81)	6 (19)	31
Total	110 (73)	41 (27)	151

TABLE VIII-3—Deaths and Case Fatality Rates within 12 Months of Birth by Number of Hospitalizations

Number of Hospitalizations	No. of Deaths	No. of Cases	% Case Fatality Rate
One	23	110	21
Two +	8	41	20
Total	31	151	21

bers of the hospitalized set were members of the seroprevalence set, 15 percent of HIV-seropositive infants born in these seven months were hospitalized with an ARD within 12 months of birth. The monthly ratios ranged from 14 to 18 percent (Table VIII-1).

The 151 ARD cases had a total of 240 hospitalizations during the 12 months of follow-up. Most (73 percent) had only one hospitalization, 16 percent had two, 5 percent had three, and 6 percent had four to eight. Cases with a diagnostic code of 795.8 had the greatest proportion of multiple admissions (Table VIII-2).

The multiple admission patterns are influenced by a high case fatality rate of 21 percent (Table VIII-3). Of the 31 deaths, 23 (74 percent) occurred during the first hospitalization. As would be expected, cases with a 042 (AIDS) diagnostic code had the highest fatality rate (40 percent), compared with 18 percent for those with a 043 or a 044 code and 9 percent for those with a 795.8 or other code.

The 240 hospitalizations of the 151 cases comprised a total of 5,772 patients days, ranging from one to 368 days per hospitalization. Nearly half of the hospitalizations had a length of stay (LOS) exceeding 10 days (Figure 1). Cases with 042 codes had the greatest median LOS, 15 days (Table VIII-4). The other four categories had similar median LOS, ranging from 7.5 to 9.0 days.

Twenty-eight percent of the cases were first hospitalized for an ARD during the birth stay or within 90 days of birth (Figure 2). More than half (56 percent) were first hospitalized within six months of birth. Fifteen cases had an ARD at the hospitalization of birth. Seven of these 15 had a diagnostic code of 795.8; all 15 had an accompanying illness, such as severe infection or drug withdrawal. The average LOS for an ARD at the hospitalization of birth was 68.6 days.

Discussion

In this study, 151 of 995 (15 percent) newborns positive for HIV antibody were hospitalized with an ARD during the first year of life. Assuming maternal transmission rates of 25 to 50 percent,[3–10,15,16] it is estimated that of the 995 HIV-positive newborns, 250–498 infants were actually HIV-infected. Thus approximately 30–60 percent of the HIV-infected infants were hospitalized with an ARD within the first year of life.

The accuracy of this estimate is difficult to assess due to problems inherent in ecologic comparisons, including migration, deaths occurring outside the hospital, underreporting or inaccurate reporting of ARD cases to SPARCS, and missing and unknown data in both data bases. To minimize the impact of migration, we only included New York State residents from both files. However, 6 percent of the ARD cases included in this study had a county of residence at first hospitalization that was not a county of residence at birth for HIV-seropositive infants born in the same month.

Undercounting of ARD cases could occur when an infant had HIV-related illness, such as a bacterial infection, but was not coded as having HIV infection. This type of underreporting was assessed among 199 adult AIDS cases identified through a one-day hospital bed census and verified through chart review. SPARCS records were identified for 188 of those cases, and 177 indicated one of the current or discontinued HIV codes (personal communication, unpublished report, Benedict I. Truman, MD, Gerald Kaufman, PhD). To assess the accuracy of pediatric AIDS reporting to SPARCS, the AIDS Surveillance Program conducted chart reviews on 56 infants reported to SPARCS with a 042 diagnostic code in 1986. Half the cases were CDC-defined AIDS, 40 percent were AIDS-related conditions or suspect AIDS, and 10 percent were either adult or duplicate cases (personal communication, unpublished report, Benedict I. Truman, MD, Gerald Kaufman, PhD).

These analyses suggest that SPARCS data capture approximately 90 percent of HIV-related hospitalizations but are not very accurate in distinguishing AIDS from

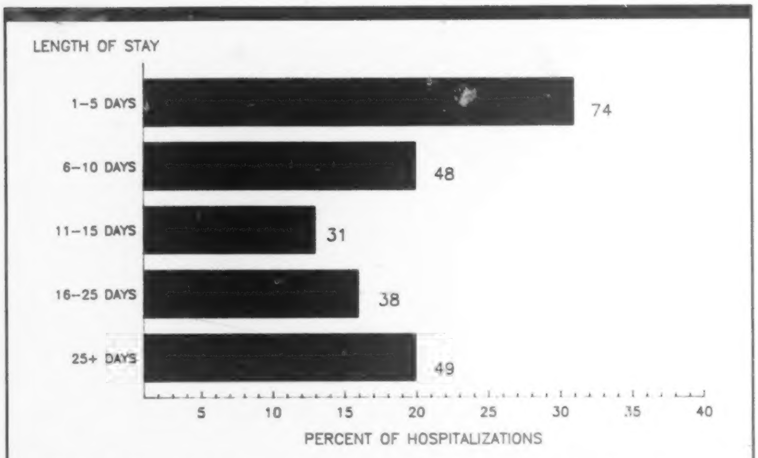

FIGURE 1—Distribution of Hospitalizations of ARD Cases by Length of Stay (in Days)

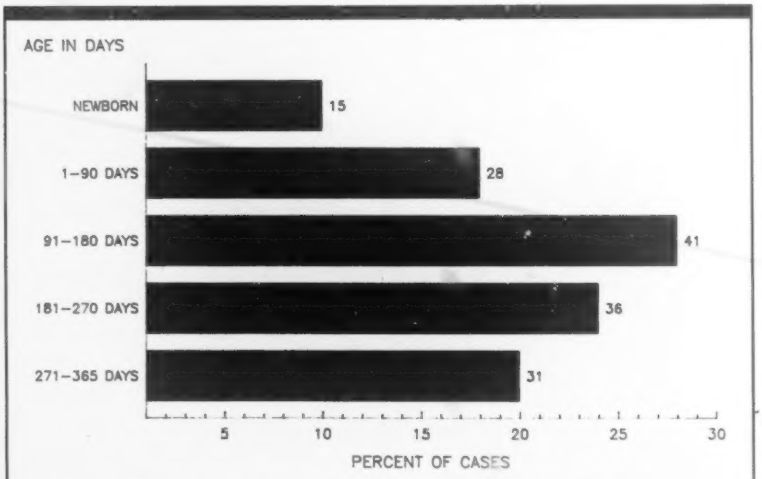

FIGURE 2—Distribution of ARD Cases by Age in Days at First Hospitalization

ARD. The latter finding was also substantiated in this study, in which nine cases switched from a 042 diagnosis to a 043, 044, or 795.8 diagnosis in subsequent hospitalizations.

Our finding that 15 percent of seropositive newborns are hospitalized with an ARD within 12 months of birth is consistent with two recent reports on pediatric AIDS.[9,10] The first study, conducted in Zaire, included HIV-seropositive infants who had lived beyond one month of age. Of the 412 infants studied, 13.4 percent had developed AIDS within 12 months of life, according to a definition based on the WHO (World Health Organization) clinical case definition of pediatric AIDS in Africa.[10] In the second study, from France, 19 (11 percent) of 177 seropositive infants developed severe forms of HIV infection within 18 months, based on the CDC classification for children.[9]

These two studies are not directly comparable with ours, because they used different study populations and case definitions. The French and Zaire studies were facility-based, whereas ours used the population-based Newborn HIV Seroprevalence Study and hospital discharge data. In addition, due to confidentiality concerns, our study could not identify HIV-seropositive or ARD cases but relied on an ecologic comparison based on month of birth. However, the similarity of results in the three studies, along with the internal consistency of month-to-month ARD/HIV ratios in this study, lend support to the accuracy of these estimates.

The results of this study are based on seven months (December 1987 through June 1988) of newborn seroprevalence data. This procedure will be conducted on an ongoing basis to project future hospital care needs of HIV-infected infants and to provide more information regarding maternal transmission and latency of HIV-related diseases in this population.

Summary

The purpose of this study was to estimate the proportion of HIV-seropositive newborns hospitalized for an AIDS-related diagnosis within 12 months of birth and to characterize these hospitalizations. The number of HIV-seropositive infants born December 1987 through June 1988 was derived from the blinded New York State Newborn HIV Seroprevalence Study. The number of infants who were born in these same months and hospitalized with an ARD within 12 months of birth was obtained from a data base containing information on all hospital discharges in New York State. Comparisons were made on the basis of month of birth.

Of infants born during the study period, 995 tested positive for HIV antibody, and 151 infants (15 percent) were hospitalized with an ARD within 12 months of birth. The 151 ARD cases had a total of 240 hospitalizations, accounting for 5,772 patient days. Fifty-six percent of the ARD cases were diagnosed within six months of birth. ☐

References

1. Hoff R, Berardi VP, Weiblen BJ, Mitchell ML, Grady GF: Seroprevalence of human immunodeficiency virus among childbearing women: Estimating by testing samples of blood from newborns. N Engl J Med 1988; 318:525–530.
2. Alford CA: Immunoglobulin determinations in the diagnosis of fetal infection. Pediatr Clin North Am 1971; 18:99–113.
3. Abrams E: The New York City Collaborative Study Group for Maternal Transmission of Human Immunodeficiency Virus (HIV). Longitudinal study of infants born to women at risk for AIDS. Abstract 7258, IV International Conference on AIDS, Stockholm, Sweden 1988; 422.
4. Scott GB, Mastrucci MT, Hutto SC, Parks WP: Mothers of infants with HIV infection: Outcome of subsequent pregnancies. Abstract THP.91, III International Conference on AIDS, Washington, DC 1987; 178.
5. Rubinstein A: Pediatric AIDS. Curr Probl Pediatr 1986; 16:361–409.
6. Minkoff H, Nandu D, Menez R, Fikrig S: Pregnancies resulting in infants with acquired immunodeficiency syndrome or AIDS-related complex: Follow-up of mothers, children, and subsequently born siblings. Obstet Gynecol 1987; 69:288–291.

TABLE VIII-4—Length of Stay for ARD Hospitalizations by Diagnosis

Diagnosis (ICD-9CM Code)	No. of Hospitalizations	Length of Stay (in Days)			
		Range	Median	Mean	SD
042	77	1-368	15.0	27.1	47.89
043	12	2-163	7.5	36.0	55.19
044	43	1-227	9.0	25.1	43.41
795.8	74	1-210	8.0	20.4	36.92
Other	34	2-205	7.5	19.4	41.23
Total	240	1-368	10.0	24.1	43.23

7. Blanche S, Rouzioux C, Veber F, *et al:* Prospective study on newborns of HIV seropositive women. Abstract TH.7.4, III International Conference on AIDS, Washington, DC 1987; 158.
8. Mendez H, Willoughby A, Hittelman J, *et al:* Human immunodeficiency virus (HIV) infection in pregnant women and their offspring. Abstract 1466, Pediatric Res 1987; 21:418A.
9. Blanche S, Rouzioux C, Guihard Moscato ML, *et al:* A prospective study of infants born to women seropositive for human immunodeficiency virus type 1. N Engl J Med 1989; 320:1643–1648.
10. Ryder RW, Nsa W, Hassig SE, *et al:* Perinatal transmission of human immunodeficiency virus type 1 to infants of seropositive women in Zaire. N Engl J Med 1989; 320:1637–1642.
11. Goetz DW, Hall SE, Harblson RW, *et al:* Pediatric acquired immunodeficiency syndrome with negative human immunodeficiency virus antibody response by enzyme-linked immunosorbent assay and Western blot. Pediatrics 1988; 81:356–359.
12. Marshall GS, Barbour SD, Plotkin SA: AIDS in a child without antibody to HIV. Lancet 1987; 1:446–447.
13. New York State Department of Health: State Planning and Research Cooperative System, New York State Department of Health, Annual Report 1985. Albany, NY: New York State Department of Health, 1985.
14. Novick LF, Glebatis DM, Stricof RL, MacCubbin PA, Lessner L, Berns DS: Newborn seroprevalence study: Methods and results. Am J Public Health 1991; 81(Suppl):15–21, Ch II.
15. Thomas PA: The New York City Department of Health: The New York City perinatal transmission study. Abstract Th.A.0.7. V International Conference on AIDS, Montreal, Canada, June 8, 1989.
16. The European Collaborative Study: Mother-to-child transmission on HIV infection. Lancet 1988; 2:1039–1043.

APPENDIX

042	Human immunodeficiency virus infection with specified conditions
043	Human immunodeficiency virus infection causing other specified conditions
044	Other human immunodeficiency virus infection
795.8	Positive serological or viral culture findings for HIV

"Other" AIDS-Related Diagnosis

279.19	Deficiency of cell-mediated immunity
279.3	Unspecified immune deficiency
136.3	*Pneumocystis Carinii Pneumonia* (PCP)
173	Other malignant neoplasm of skin (for Kaposi's Sarcoma, KS)
117.5	Cryptococcosis
321.0	Cryptococcal meningitis
031	Mycobacterial Disease (non-TB)
130.0	Toxo meningo encephalitis
54.7	Herpes simplex w/other complications
200.0	Reticulosarcoma
200.8	Other named variants, lymphomas
516.8	Interstitial pneumonia
112.4	Candidiasis, lung
46.3	P.M. Leucoeneph
115	Histo

IX. HIV Seroprevalence in a Facility for Runaway and Homeless Adolescents

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Introduction

Despite the clinical and epidemiologic importance of HIV infection among adolescents, our knowledge of the extent of infection in this population in New York State has been limited, relying on AIDS case reports and data on military recruits. As of December 31, 1989, a total of 67 cases of AIDS in individuals 13–19 years old had been reported statewide, 45 males and 22 females. Their risk factors were reported as follows: 21 were homosexual/bisexual males, 12 were intravenous drug users, three were homosexual intravenous drug users, 10 were heterosexual contacts of persons at risk, 14 received blood products, three were foreign-born, and risk factors were not determined for four.

Military recruit data for New York State residents from October 1985 through December 1988 revealed a seroprevalence of 0.06 percent for persons 17–19 years old (communication from Centers for Disease Control; Quarterly Data Report on HIV in Civilian Applicants for Military Service, June 22, 1989). Thirty males and eight females were identified as seropositive for HIV.

Due to this limited availability of information on adolescents, a seroprevalence study was initiated in Covenant House New York, a facility serving runaway and homeless youth in New York City. Although this population is not representative of adolescents in general, it was selected as a sentinel group of high-risk youth. The study was designed to determine HIV seroprevalence among adolescents receiving health care at the facility, to ascertain selected demographic characteristics (age, race/ethnicity, gender), to assess trends in seroprevalence over time, and to determine the risk factors associated with HIV infection in this population.

Background

Covenant House New York predominantly serves adolescents under the age of 21. It consists of a crisis center, which can accommodate more than 300 individuals a night; a drug treatment program for 26 residents, with a 30-day average stay; a mother-child shelter; an outreach program (vans that go out at night); and a long-term transitional living program.

A recent survey[1] of 168 Covenant House adolescents revealed the following sociologic characteristics:

- The average length of time away from home was 90 weeks.
- Alcohol was used by 80 percent, marijuana by 68 percent, cocaine by 48 percent, and crack by 38 percent.
- Intravenous drug use was low (6 percent).
- Nearly all (91 percent) were sexually active, with an average of 2.8 sexual partners weekly (range, one to 20).
- Twenty-nine percent admitted to having exchanged sex for food, money, shelter, or drugs.
- Most (62 percent) reported having been arrested.
- Almost half (46 percent) reported having been treated as outpatients for psychiatric problems, and 18 percent had received inpatient psychiatric treatment.

Approximately 50–60 percent of the adolescents seeking shelter in Covenant House New York are seen in the health services department. Individuals are referred for health services immediately if they are on medication, placed in one of the group homes, a mother-infant pair, or deemed to have a health problem on intake. All others are referred if they remain in the facility for 24–48 hours. At the first visit a complete health assessment is performed, which includes a physical examination, urinalysis, complete blood count, and serologic test for syphilis.

TABLE IX-1—HIV Seroprevalence by Quarter among Runaway and Homeless Adolescents, October 1987–December 1989

Quarter	Number Positive	Number Tested	Percent Positive
4th 1987	22	280	7.9
1st 1988	14	315	4.4
2nd 1988	18	254	7.1
3rd 1988	20	292	6.9
4th 1988	11	250	4.4
1st 1989	16	300	5.3
2nd 1989	13	245	5.3
3rd 1989	20	415	4.8
4th 1989	8	316	2.5
Total	142	2,667	5.3

Methods

Study subjects were limited to Covenant House New York clients undergoing an initial medical evaluation between October 1, 1987 and December 31, 1989. Clients seen for annual or follow-up visits were excluded to avoid repeat testing of the same individuals over time. The study was limited to persons 15–20 years old, because few individuals under the age 15 or over age 20 visit the clinic and we wanted to be able to assure anonymity.

If a client met the inclusion criteria, an HIV seroprevalence study test requisition (SSTR) form was completed by the clinic staff. The completed form contained the adolescent's age, race or ethnicity, gender, and most recent county and zip code of residence, as well as the month and year of specimen collection. No personal identifier (e.g. name, previous street address, or medical record number) was recorded.

The SSTR form was held with the specimen until all routine laboratory tests were completed. If serum was left over, the laboratorian would pipette at least 0.5 ml into a tube labeled with the blind seroprevalence number and send the serum with the SSTR form to the New York State Department of Health's Wadsworth Center for Laboratories and Research for HIV testing.[2] No additional blood was drawn for this study. If there was not a sufficient quantity of blood left over, the demographic data alone were sent to the Department and entered into the data base.

In May 1988 a risk-assessment component was added to the study design. Information routinely obtained by the clinic staff and documented in the medical record was abstracted onto a separate risk assessment form with the blind seroprevalence number. This information included, where applicable, the gender of sex partners; the history of intravenous drug use (heroin or cocaine), crack use, prostitution, blood transfusion, and/or sexually transmitted disease; and the sex partners' history of drug use, homosexual/bisexual activity, or HIV status.

The information recorded on the SSTR form, including the HIV test result, was merged with the risk-assessment information and analyzed using computer software from SAS Institute, Inc; BMDP Statistical Software, Inc; the Centers for Disease Control[3]; and Epistat Services.[4]

Overall HIV seroprevalence rates were calculated by quarter-year of clinic visit. Chi-square tests for trend were applied to the data.[4]

Seroprevalence rates were calculated by gender, year of age, and for three racial/ethnic categories: non-Hispanic Whites, non-Hispanic Blacks, and Hispanics. The effects of the various demographic characteristics were assessed by multiple regression analysis after controlling for potential confounding effects of the others.

Univariate analysis for risk factors associated with HIV infection was performed with odds ratios and confidence intervals.[5] Mutually exclusive categories were established in the following hierarchical order: intravenous-drug-using homosexual/bisexual males, homosexual/bisexual males, intravenous drug users, blood transfusion recipients, persons with sex partners known or at risk for infection, persons with a history of sexually transmitted disease, prostitutes, and crack users. Individuals reporting none of these risk factors formed the reference group.

Demographic characteristics and/or risk factors independently associated with HIV infection were determined by multiple logistic regression after controlling for the effects of the others. Age, race/ethnicity, and risk factors derived from the univariate analysis were forced into the model. Adjusted odds ratios and confidence intervals were derived from the parameter estimates and standard errors.

Results

Over the 27-month study period, 2,667 specimens were analyzed for HIV antibody, and 142 (5.3 percent) were found to be positive. Five specimens were not tested due to insufficient serum, and eight equivocals were eliminated from the analysis.

The HIV seroprevalence rates by quarter are presented in Table IX-1. There was a tendency toward a decrease in seroprevalence over time, but the decline was not sufficient overall ($p = 0.27$) or for any subset of the population (males, females, non-Hispanic Whites, non-Hispanic Blacks, Hispanics, 15–16-year-olds, 17–18-year-olds, or 19–20-year-olds). A tendency toward a decline approaching statistical significance was found for females ($p = 0.06$), Blacks ($p = 0.08$), and 19–20-year-olds ($p = 0.09$).

Seroprevalence rates by age, race/ethnicity, and gender are presented in Table IX-2. The rates increased from 1.3 percent for 15-year-olds (not shown) to 8.6 percent for 20-year-olds. Hispanics had the highest seroprevalence rate, followed by non-Hispanic Whites and non-Hispanic Blacks. For males the rate was 6.0 percent; for females, 4.2 percent. However, after controlling for each of the other demographic characteristics, age was the only variable found to be associated with HIV seropositivity.

Between May 1988 and December 1989 risk-assessment forms were submitted on 1,758 individuals, 1,039 males and 719 females. The unadjusted HIV seroprevalence rates and univariate risk-factor analyses are presented in Table IX-3. HIV infection in males was associated with intravenous drug use, homosexual/bisexual activity, history of a sexually transmitted disease, prostitution, and crack use. HIV infection in females was associated with history of another sexually transmitted disease.

The results of the multivariate risk-assessment analyses are summarized in Table IX-4. After controlling for the confounding effects of the other variables listed, HIV infection in male adolescents was found to be associated with the following independent risk factors in declining order: homosexual/bisexual activity, prostitution, having had another sexually

TABLE IX-2—HIV Seroprevalence Rates by Demographic Characteristics among Runaway and Homeless Adolescents, October 1987–December 1989

Demographic Characteristics	Number Positive*	Number Tested*	Percent Positive	Odds Ratio**	95% Confidence Interval**
Age (years)					
15–16***	7	312	2.2	1.0	
17	10	340	2.9	1.1	0.4, 3.1
18	20	591	3.4	1.3	0.6, 3.2
19	49	750	6.5	2.7	1.2, 6.0
20	56	653	8.6	3.7	1.7, 8.3
Race/Ethnicity					
Non-Hispanic White	24	399	6.0	1.0	
Non-Hispanic Black	67	1,448	4.6	0.7	0.4, 1.2
Hispanic	47	688	6.8	1.1	0.7, 1.5
Gender					
Female	44	1,037	4.2	1.0	
Male	97	1,611	6.0	1.2	0.8, 1.8

*Differences in the total numbers found positive and the total numbers tested for each parameter reflect missing demographic data on the SSTR forms.

**Odds ratios and confidence intervals after controlling for the other demographic characteristics.

***Combined for logistic regression analysis due to small cell sizes.

TABLE IX-3—Univariate Analysis of Risk Factors for HIV Infection by Gender among Runaway and Homeless Adolescents, May 1988–September 1989

Hierarchical Risk Factor	Number Positive	Number Tested	Percent Positive	Odds Ratio	95% Confidence Interval
Males					
IVDU and Homosexual or Bisexual	4	12	33.3	47.5	8.0, 243.0
Homosexual or Bisexual	27	109	24.8	31.2	12.1, 94.7
IVDU	3	32	9.4	9.8	1.5, 48.4
Blood Transfusion	1	9	11.1	11.9	0.2, 116.6
Sex Partner at Risk	1	11	9.1	9.5	0.2, 89.9
STD	4	69	5.8	5.9	1.2, 25.3
Prostitution	2	7	28.6	38.0	2.9, 290.4
Crack Use	11	214	5.1	5.2	1.7, 17.1
No Risk Indicated	6	576	1.2	1.0	
Females					
IVDU	2	16	12.5	8.5	0.8, 47.9
Blood Transfusion	0	3	0.0	0.0	0.0, 158.7
Sex Partner at Risk	1	8	12.5	8.5	0.2, 79.9
STD	8	124	6.4	4.1	1.3, 12.8
Prostitution	2	16	12.5	8.5	0.8, 47.9
Crack Use	2	69	2.9	1.8	0.2, 9.1
No Risk Indicated	8	483	1.7	1.0	

Abbreviations: IVDU, intravenous drug use; STD, sexually transmitted disease (other than HIV infection).

transmitted disease, and crack use. For females no risk factors were independently associated with HIV infection, although there was a tendency toward an association for prostitution and having had another sexually transmitted disease.

Discussion

This seroprevalence study of a sentinel group of runaway and homeless adolescents disclosed an alarming rate of HIV infection. More than 5 percent were seropositive. Although not representative of adolescents in general, they demonstrate the magnitude of the problem for a large and definable group at one end of the spectrum. These findings have implications, not only for clients of this facility, but also for other groups who interact with runaways and homeless and are consequently at risk.

These homeless youths come from the streets of New York City; their environment is characterized by drug use, prostitution, poverty, and crime. The seroprevalence data indicate an association between HIV infection and prostitution, history of other sexually transmitted diseases, and crack use, in addition to other, previously defined risk factors. Exchanging sex for food, money, shelter, or drugs with multiple sexual partners, some of whom are infected, is the most likely explanation for HIV transmission in this group of adolescents.

Since we were limited to obtaining risk information that was documented in the medical record during the standard course of medical practice, the data probably do not reflect the full incidence of these risk behaviors and should be interpreted with caution. Data provided by Margetson and Lipman[1] suggest that prostitution and crack use were underreported in our study.

The adolescents in this study are highly susceptible hosts living in a hazardous environment. They are regularly exposed to infectious agents, including HIV. Host factors include high levels of sexual activity, unprotected sexual intercourse, and homosexual/bisexual activity in males. Environmental factors include poverty and the necessity to support oneself and obtain drugs in an environment where drugs, prostitution, sex, and infection are commonplace and linked. Prevention activities face the challenge of inducing change, not only in the individuals, but in the external factors that facilitate exposure.

Prevention initiatives must start earlier than the adolescent period if a satisfactory impact is to be attained. All schools in New York State must provide basic HIV education. Regional training coordinators from the New York State Education Department are helping to develop and implement these programs.

The New York State Department of Health, using state and federal funds, has contracted with a variety of community-based organizations to provide HIV education, counseling, and testing services as well as special living arrangements or referrals, basic needs (food, clothing, shelter, etc.), psychosocial support, and medical services to high-risk adolescents.

Summary

In October 1987, the New York State Department of Health initiated a study to determine the prevalence of antibody to HIV in clients of a facility for runaway and homeless adolescents in New York City. A risk-assessment component was added in May 1988. As of December 1989, a total of

TABLE IX-4—Multivariate Analysis of Factors Associated with HIV Infection among Runaway and Homeless Adolescents, May 1988–December 1989

Demographic Characteristic or Risk Factor	Odds Ratio (95% Confidence Interval)*	
	Males	Females
Race/Ethnicity		
Non-Hispanic White	1.0	1.0
Non-Hispanic Black	1.0 (0.4, 2.6)	1.1 (0.3, 3.8)
Hispanic	2.0 (0.8, 5.2)	0.6 (0.1, 3.3)
Homosexual/Bisexual Male	6.1 (2.8, 12.9)	—
Intravenous Drug Use	0.9 (0.3, 2.9)	1.9 (0.3, 11.7)
Crack Use	2.3 (1.2, 4.3)	1.3 (0.4, 3.9)
History of STD**	2.4 (1.1, 5.0)	2.3 (0.9, 6.2)
Prostitution	2.8 (1.0, 7.5)	4.0 (0.9, 18.3)

*After controlling for age.
**Sexually transmitted disease (other than HIV infection).

2,667 adolescents had been tested, and 142 (5.3 percent) were found to be HIV-seropositive (males 6.0 percent, females 4.2 percent). The seroprevalence rate increased from 1.3 percent for 15-year-olds to 8.6 percent for 20-year-olds. Hispanics had the highest seroprevalence rate (6.8 percent), followed by non-Hispanic Whites (6.0 percent) and non-Hispanic Blacks (4.6 percent). HIV seropositivity was associated with intravenous drug use, male homosexual/bisexual activity, prostitution, and history of another sexually transmitted disease. The alarmingly high prevalence of HIV infection in this selected population illustrates the immediate need for prevention programs for adolescents. □

Acknowledgments

The authors gratefully acknowledge the contribution of the clinical, nursing, and laboratory staff of Covenant House New York, particularly Olga Hernandez and Constance Camacho. Statistical consultation and support was provided by Hwa-Gan Chang. This study was partially funded by the Centers for Disease Control.

References

1. Margetson N, Lipman C: Children at risk: The impact of poverty, the family and the street on homeless and runaway youth in New York City. Delivered at the National Symposium on Youth Victimization, Atlanta, GA, April 27, 1990.
2. Stricof RL, Nattell TC, Novick LF: HIV seroprevalence in clients of sentinel family planning clinics. Am J Public Health 1991; 81(Suppl):41–45, Ch VII.
3. Dean JA, Dean AG, Burton A, Dieker R: Epi Info, Version 3. Atlanta, GA: Centers for Disease Control, January 1988.
4. Gustafson TL: True Epistat, Third Ed. Richardson, TX: Epistat Services, 1989.
5. Mehta CR, Patel NR, Gray R: Computing an exact confidence interval for the common odds ratio in several 2 × 2 contingency tables. J Am Stat Assn 1985; 80:969–973.

X. The AIDS Epidemic in New York State

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Introduction

Surveillance for cases of acquired immunodeficiency syndrome (AIDS) has provided important information for characterizing and controlling the epidemic. Even before the human immunodeficiency virus (HIV) had been identified in 1983 and 1984,[1,2] AIDS surveillance demonstrated that gay men and intravenous drug users (IVDUs) bore a disproportionate share of the burden of disease.[3] Surveillance data provided the first information about the modes of transmission of HIV and were thus the basis for early recommendations to limit its spread.[4]

New York State reported some of the first AIDS cases and has the highest number of cases in the nation, mostly from New York City. This report describes the AIDS epidemic statewide and compares it with the epidemic in the rest of the nation. It also compares data from the state's Newborn HIV Seroprevalence Study with AIDS surveillance data for women.

Methods

Both the New York State Department of Health (DOH) and New York City DOH maintain active surveillance for AIDS cases, including regular solicitation of reports from hospitals. New York City DOH staff investigate all cases reported within the city; New York State DOH staff investigate cases in New York State exclusive of New York City. Once a suspect case is identified, trained surveillance staff review the patient's medical records to collect demographic, risk-factor, and clinical information. Vital status is regularly updated by systematic review of death certificate reports and by encouraging hospitals to report deaths to surveillance staff.

Throughout the epidemic, all suspect cases have been evaluated according to the national surveillance case definition, which has been expanded twice, in 1985 and 1987.[3,5,6] Each case was evaluated by the criteria current when the case was investigated. Cases meeting more than one definition were classified hierarchically according to the earliest definition that they met.

Special case ascertainment studies indicate that 75-85 percent of diagnosed AIDS cases in New York City are eventually reported,[7] as are more than 95 percent of patients discharged with AIDS-related diagnoses in New York State exclusive of New York City.[8]

Both the State and New York City DOH report their confirmed cases independently, without identifying information, to the Centers for Disease Control (CDC) for national surveillance. The New York State DOH also maintains a statewide computerized AIDS registry in which the state and city case files are merged.

For comparison with the statewide case data, the number of cases in the rest of the United States was determined from summary national AIDS surveillance data reported to CDC through November 1989.[9] For each subgroup comparison the number of cases reported statewide was subtracted from the corresponding national total. To ensure comparability between the statewide and national data, we selected all cases registered in New York State exclusive of New York City as of October 31, 1989, and all cases registered in New York City as of November 17, 1989—the final dates of data transfer for the national surveillance report.

Geographic classification of persons with AIDS was based on their residence at the time they met the national surveillance case definition, except for cases outside New York City before July 1989, where we used the residence at onset of symptoms. Cases were also classified as adults/

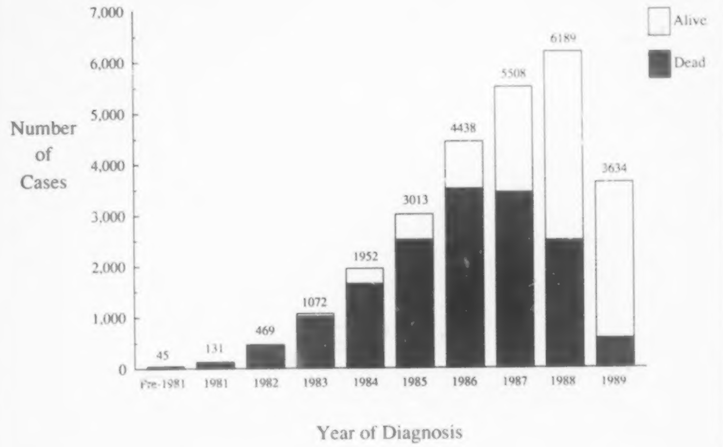


FIGURE 1—Number of AIDS cases (N = 26,576) by Vital Status and Year of Diagnosis, New York State

NOTE: A total of 125 cases with unknown vital status were excluded. Data for 1989 are incomplete due to the delay in case reporting.

adolescents (>12 years old) or children (<13 years old) and by hierarchical exposure groups, according to the CDC system.[9]

As a measure of HIV transmission among adolescents, data for persons 20–24 years old were analyzed. Adults in this age group are likely to have been infected during adolescence because of the long median incubation period for AIDS, which studies suggest is about 10 years.[10–12] Cases among teenagers were not included in this analysis, since their infections may have occurred before adolescence.

AIDS incidence rates were calculated with official New York State population projections by age, sex, and geographic region for 1985.[13] For race-specific population estimates, we applied the racial distribution from the 1980 Census to the 1985 projections, since race-specific projections are not available.

For comparison with results of the Newborn HIV Seroprevalence Study,[14] we used the incidence of AIDS per 100,000 women of childbearing age (15–49 years) during 1988. From the Newborn HIV Seroprevalence Study, we used data for all births to mothers in this age group in 1988. The data were stratified by age, race/ethnicity, and geographic regions, according to urbanization and proximity to New York City, as described elsewhere.[14]

Results

Cases by Type of Definition

A total of 26,576 cases of AIDS were reported statewide by the cutoff dates for this report, accounting for 23 percent of the 115,158 cases reported nationally. Of these cases, 22,186 (83 percent) met the pre-1985 national surveillance case definition,[3] 592 (2 percent) met the 1985 definition,[5] and 3,798 (14 percent) met the expanded 1987 definition.[6] Of the 1987 definition cases, 47 percent had indicator diseases that were diagnosed definitively; the rest were diagnosed presumptively. Of 9,847 cases diagnosed in 1988 and 1989, 29 percent met only the 1987 case definition (13 percent definitive, 16 percent presumptive). Of the total 26,576 cases, 16,163 (61 percent) had *Pneumocystis carinii* pneumonia, and 3,505 (13 percent) had Kaposi's sarcoma.

Incidence and Mortality

The number of AIDS cases diagnosed statewide has increased every year through 1988 (Figure 1). An estimated 90 percent of cases that are ultimately reported are included in the registry by 11 months after diagnosis (Dr. Lawrence Lessner, New York State Department of Health, personal communication). Because of this delay in reporting, the number of cases for 1989 is expected to exceed that for 1988 when reporting is complete.[15]

Of the 26,576 reported cases, 17,531 (66 percent) are known to have died as of the cutoff dates for this study. Thirteen percent of all cases died within one month of diagnosis. The case fatality rate is highest for cases with the longest follow-up: 90 percent of cases diagnosed before 1985. Of patients diagnosed before 1988, 80 percent have died (median survival time, 10.3 months).

Geographic Distribution

New York City accounts for 22,231 (84 percent) of all statewide cases, although the percentage has dropped as the epidemic spread. Another 3,250 cases (12 percent) were reported among the general population from New York State exclusive of New York City and 1,095 (4 percent) among prisoners in the state correctional system.

The geographic distribution of cases in quartiles is shown in Figure 2. Cases among prisoners are excluded, because accurate information on their counties of residence was not available. Although cases are widely distributed throughout the state, they tend to concentrate in counties with major metropolitan areas (e.g., Albany, Erie, Monroe, and Onondaga), as well as in New York City and nearby counties.

Gender

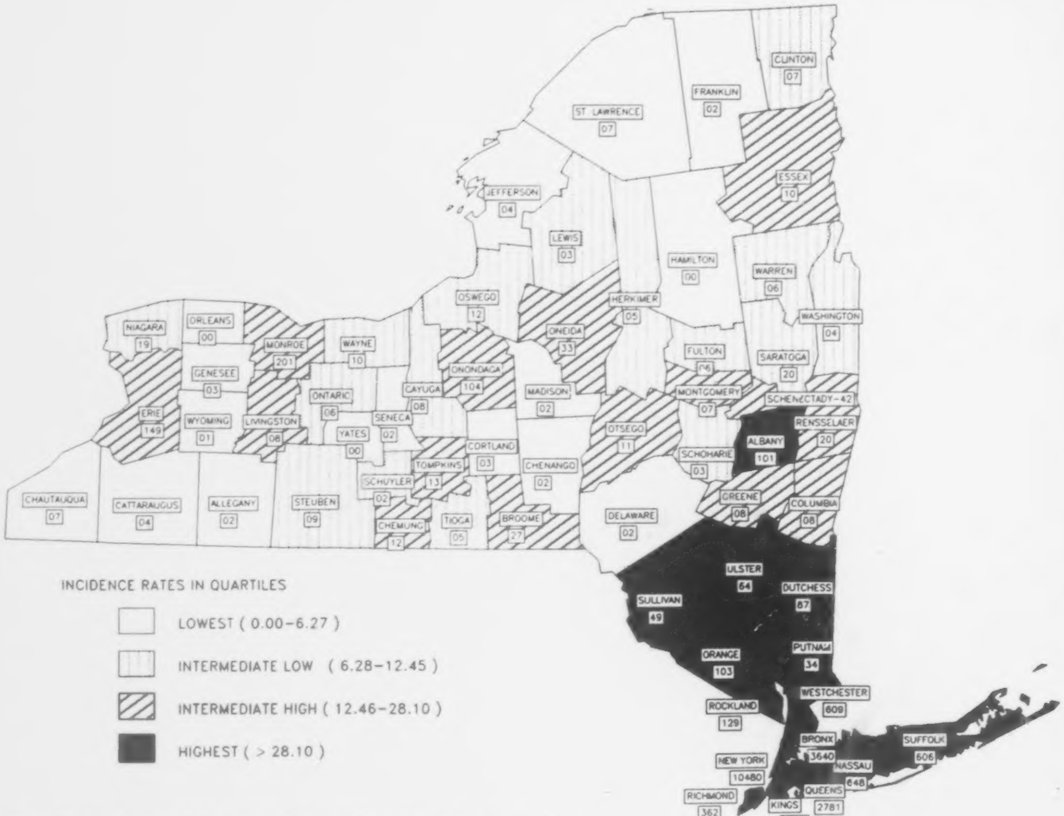
Males account for 22,482 (87 percent) of the statewide adult/adolescent cases (Table X-1). There is no difference in gender distribution between cases in New York City and New York State exclusive of New York City. The male case predominance reflects the large number of cases among men with a history of homosexual/bisexual contact. However, this percentage is lower than that for the rest of the United States, because New York State has a disproportionately high number of women who contracted AIDS by intravenous drug use.

Among children there is a slightly higher proportion of cases among males, but the gender distribution differs little from the rest of the United States (Table X-2).

Age

The age distribution of adult/adolescent cases statewide is similar to that in the rest of the United States, with nearly 90 percent of cases occurring among persons 20–49 years old (Table X-1).

Among children, New York State has 604 reported cases statewide and accounts for 31 percent of all such cases in the United States (Table X-2). Of these pediatric patients, 534 (88 percent) were


CLINTON 07
FRANKLIN 02
ST LAWRENCE 07
ESSEX 10
JEFFERSON 04
LEWIS 03
HAMILTON 00
WARREN 06
OSWEGO 12
HERKIMER 05
WASHINGTON 04
NIAGARA 19
ORLEANS 00
MONROE 201
WAYNE 10
ONEIDA 33
GENESEE 03
ONONDAGA 104
FULTON 06
SARATOGA 20
CAYUGA 08
MADISON 02
MONTGOMERY 07
SCHENECTADY-42
ERIE 149
WYOMING 01
LIVINGSTON 08
ONTARIO 06
SENECA 02
YATES 00
CORTLAND 03
OTSEGO 11
ALBANY 101
RENSSELAER 20
TOMPKINS 13
SCHUYLER 02
CHENANGO 02
SCHOHARIE 03
GREENE 08
COLUMBIA 08
CHAUTAUQUA 07
CATTARAUGUS 04
ALLEGANY 02
STEUBEN 09
CHEMUNG 12
TIOGA 05
BROOME 27
DELAWARE 02
ULSTER 64
DUTCHESS 87
SULLIVAN 49
ORANGE 103
PUTNAM 34
WESTCHESTER 609
ROCKLAND 129
SUFFOLK 606
BRONX 3640
NASSAU 648
NEW YORK 10480
QUEENS 2781
RICHMOND 362
KINGS 4852
INCIDENCE RATES IN QUARTILES
☐ LOWEST (0.00–6.27)
▥ INTERMEDIATE LOW (6.28–12.45)
▨ INTERMEDIATE HIGH (12.46–28.10)
■ HIGHEST (> 28.10)

[1] BASED ON NYS DEPARTMENT OF COMMERCE 1985 OFFICIAL POPULATION PROJECTIONS

FIGURE 2—Cumulative number of AIDS Cases and Incidence per 100,000 Population since the beginning of the Epidemic, by County or Borough of Residence, New York State

NOTE: Cases among New York State prison inmates were excluded. Cumulative incidence was based on New York State Department of Commerce population projections for 1985.

residents of New York City. Compared with the rest of the United States a greater proportion of cases occurred among children under five years old, reflecting the greater proportion of children infected perinatally in this state.

Race/Ethnicity

Whereas Blacks and Hispanics account for 37 percent of adult/adolescent cases in the rest of the United States, they account for nearly 60 percent of such cases statewide (Table X-1). This difference reflects the large percentage of Black and Hispanic cases in New York City (62 percent) and in state prisons (85 percent). In New York State exclusive of New York City, Blacks and Hispanics account for 37 percent of cases.

The statewide cumulative incidence rate (per 100,000 population) from the beginning of the epidemic among adult/adolescent males is several times higher among Blacks (901) and Hispanics (1,029) than among Whites (183) (Table X-3). For females, the racial/ethnic disparity is even greater (Table X-4).

Of children with AIDS, nearly 90 percent statewide are Black or Hispanic, compared to 72 percent in the rest of the United States (Table X-2).

Exposure Groups

The AIDS epidemic among adults and adolescents has primarily affected two exposure groups: men with a history of homosexual/bisexual contact, and IVDUs. The epidemic curve for gay men has leveled off or declined in recent years (Figure 3).

In 1988, for the first time, more cases were diagnosed among IVDUs than among gay men statewide. The percentage of cases among IVDUs is greater statewide (39 percent) than in the rest of the United States (16 percent) (Table X-1). Of the 1,095 prisoners with AIDS, 957 (87 percent) gave a history of intravenous drug use without homosexual/bisexual contact. Even when prisoners are excluded, the percentage of cases with a history of intravenous drug use is still higher in New York City (36 percent) and in New York State exclusive of New York City (35 percent) than in the rest of the United States (16 percent). The percentage of

TABLE X-1—Distribution of AIDS Cases among Adults/Adolescents (Age 13 and Older) in New York State and in the Rest of the United States

Demographic Variables	New York State		United States (Excluding New York State)	
	Number	(%)	Number	(%)
Sex				
Males	22,482	(86.6)	80,360	(92.0)
Females	3,490	(13.4)	6,879	(8.0)
Age Group (years)				
13–19	65	(0.3)	382	(0.4)
20–29	4,568	(17.6)	18,930	(21.7)
30–39	12,737	(49.0)	40,437	(46.4)
40–49	6,010	(23.1)	18,337	(21.0)
50+	2,592	(10.0)	9,157	(10.5)
Race/Ethnicity				
White	10,185	(39.2)	54,008	(61.9)
Black	8,729	(33.6)	21,758	(24.9)
Hispanic	6,753	(26.0)	10,655	(12.2)
Other	163	(0.6)	685	(0.8)
Unknown	142	(0.5)	133	(0.2)
Exposure Category				
Male Homosexual/Bisexual Contact	11,995	(46.2)	56,572	(64.8)
Intravenous (IV) Drug Use	10,120	(39.0)	13,602	(15.6)
Male Homosexual/Bisexual Contact and IV Drug Use	1,137	(4.4)	6,786	(7.8)
Hemophilia/Coagulation Disorder	61	(0.2)	986	(1.1)
Heterosexual Contact	1,390	(5.4)	4,067	(4.7)
Sex with Person at Risk for AIDS	903	(3.5)	2,951	(3.4)
Born in Pattern II Country*	487	(1.9)	1,116	(1.3)
Receipt of Blood Transfusion, Blood Components, or Tissue	267	(1.0)	2,501	(2.9)
Other/Undetermined	1,002	(3.9)	2,725	(3.1)

*Includes persons presumed to have become HIV-infected through heterosexual contact because they were born in countries where heterosexual transmission is believed to be the major mode of transmission.[9]

cases among non-IVDU men with a history of homosexual/bisexual contact was similar in New York City (49 percent) and New York State exclusive of New York City (44 percent).

The third most common exposure group is persons at risk for AIDS because of heterosexual contact. A slightly higher percentage of cases is in this category statewide than in the rest of the United States (5.4 vs 4.7 percent) (Table X-1). This category contains two subgroups: persons who report heterosexual contact with a person at risk for AIDS, and persons who are presumed to have become HIV-infected through heterosexual contact because they were born in countries where heterosexual transmission is believed to be the major mode of transmission, the so-called Pattern II countries.[9] Of the 1,390 statewide cases of heterosexual contact, 903 (65 percent) reported contact, and 487 (35 percent) were from Pattern II countries. Of the 903 cases with reported contact, 757 (84 percent) reported having sex with an IVDU.

The distribution of exposure categories varied by race/ethnicity and gender. Of all cases among White males, 78 percent were attributed solely to homosexual/bisexual contact, in contrast to 35 percent of Black and Hispanic males (Table X-3). Intravenous drug use was the leading exposure category for both Black (49 percent) and Hispanic (55 percent) males. Among females there was less variation, although heterosexual contact accounted for a larger percentage of cases among Blacks (27 percent) and Hispanics (34 percent) than among Whites (23 percent) (Table X-4). Overall the leading exposure categories for females were intravenous drug use (62 percent) and heterosexual contact with a person at risk for AIDS (25 percent). Of the 864 females in the latter group, 729 (84 percent) reported having sex with an IVDU.

Statewide, 91 percent of children with AIDS were classified as acquiring their infection from their mothers (perinatally), compared with 76 percent in the rest of the United States (Table X-2). This difference is largely due to the majority (59 percent) of pediatric cases statewide whose mothers used intravenous drugs.

AIDS among Young Adults

Statewide 867 AIDS cases were reported among persons 20–24 years old, of whom 251 (29 percent) were White, 298 (34 percent) were Black, and 309 (36 percent) were Hispanic; the remaining nine were of other or unknown race/ethnicity. The leading exposure groups for these cases were male homosexual/bisexual contact, 403 (46 percent); intravenous drug use, 248 (29 percent); male homosexual/bisexual contact and intravenous drug use, 40 (5 percent); heterosexual contact with an intravenous drug user, 64 (7 percent); and born in a Pattern II country, 29 (3 percent). The remaining 83 cases were attributed to other exposure categories.

Comparison of AIDS Data with Newborn HIV Seroprevalence

Two measures of HIV infection among women are shown in Table X-5: the incidence of AIDS per 100,000 women in 1988, and the rate of HIV seropositivity among women giving birth in 1988, based on data from the Newborn HIV Seroprevalence Study. The absolute rates of these measures varied because of the different events they reflect: onset of severe HIV disease and prevalence of infection among women giving birth. However, both measures showed comparable trends. The highest rates occurred among persons 20–39 years old, Blacks, Hispanics, and women residing in New York City. Both rates declined with distance from New York City. The only disproportionate trend was among persons 15–19 years old: a disproportionately lower rate of AIDS per 100,000 women relative to the older groups, compared with the HIV seropositivity rates from the newborn study.

Discussion

AIDS has become a major cause of morbidity and mortality statewide. It is the leading cause of death among men 30–49 years old and women 20–39 years old in New York City, as well as among persons 30–39 years old in minority racial/ethnic groups in New York State exclusive of New York City.[15]

Statewide the effect of the AIDS epidemic among IVDUs is apparent in its increasing toll on racial minorities, women, and children. Transmission of HIV by contaminated needles has disproportionately affected Blacks and Hispanics. Both intravenous drug use and heterosexual transmission from IVDUs have caused a greater proportion of cases among women here than in the rest of the nation, and these women have transmitted HIV perinatally to a large number of children.

AIDS surveillance data have complemented the information provided by the HIV seroprevalence studies. Whereas seroprevalence studies provide timely information on the number, geographic distribution, and changing characteristics of HIV infected persons, information from AIDS surveillance provides detailed information on each reported case since the beginning of the epidemic. AIDS surveillance data show patterns similar to those from the Newborn HIV Seroprevalence Study except for one age-group difference. Where the Newborn Seroprevalence Study shows an alarming rate of HIV infection among women 15–19 years old, surveillance data show that they have a low incidence of AIDS. This finding appears to reflect the long incubation period for AIDS. AIDS case data for persons 20–24 years old statewide suggest that HIV transmission among adolescents has occurred more frequently among Blacks and Hispanics than among Whites and that sexual contact (both homosexual and heterosexual) has been the leading risk behavior.

TABLE X-2—Distribution of AIDS Cases among Children (Age 12 and Under) in New York State and in the Rest of the United States

	New York State		United States (Excluding New York State)	
Demographic Variables	Number	(%)	Number	(%)
Sex				
Males	320	(53.0)	740	(55.1)
Females	284	(47.0)	603	(44.9)
Age Group (years)				
<5	531	(87.9)	1,071	(79.7)
5–12	73	(12.1)	272	(20.3)
Race/Ethnicity				
White	62	(10.3)	363	(27.0)
Black	334	(55.3)	693	(51.6)
Hispanic	205	(33.9)	272	(20.3)
Other	1	(0.2)	12	(0.9)
Unknown	2	(0.3)	3	(0.2)
Exposure Category				
Mother at Risk for AIDS	550	(91.1)	1,022	(76.1)
Intravenous (IV) Drug Use	359	(59.4)	449	(33.4)
Sex with IV Drug User	112	(18.5)	210	(15.6)
Sex with Other Person at Risk for AIDS/HIV	17	(2.8)	104	(7.7)
Born in Pattern II Country*	47	(7.8)	120	(8.9)
Receipt of Blood Transfusion, Blood Components, or Tissue	3	(0.5)	30	(2.2)
Has HIV Infection, Risk Not Specified	12	(2.0)	109	(8.1)
Receipt of Blood Transfusion, Blood Components, or Tissue	19	(3.1)	189	(14.1)
Hemophilia/Coagulation Disorder	4	(0.7)	100	(7.5)
Undetermined	31	(5.1)	32	(2.4)

*See Table 1.

TABLE X-3—AIDS Cases among Male Adults/Adolescents by Exposure Category and Race/Ethnicity, and Cumulative Incidence per 100,000 Males, Age 15 Years and Older, in New York State

	White		Black		Hispanic		Total*	
Exposure Category	Number	(%)	Number	(%)	Number	(%)	Number	(%)
Male Homosexual/Bisexual Contact	7,428	(77.6)	2,408	(34.8)	1,986	(34.8)	11,994	(53.4)
Intravenous (IV) Drug Use	1,365	(14.3)	3,418	(49.3)	3,129	(54.8)	7,965	(35.4)
Male Homosexual/Bisexual Contact and IV Drug Use	391	(4.1)	423	(6.1)	317	(5.6)	1,137	(5.1)
Hemophilia/Coagulation Disorder	45	(0.5)	4	(0.1)	7	(0.1)	56	(0.2)
Heterosexual Contact	17	(0.2)	375	(5.4)	4	(0.1)	398	(1.8)
Sex with Person at Risk for AIDS	17	(0.2)	17	(0.2)	4	(0.1)	39	(0.2)
Born in Pattern II Country**	0	(0)	358	(5.2)	0	(0)	359	(1.6)
Receipt of Blood Transfusion, Blood Components, or Tissue	119	(1.2)	25	(0.4)	20	(0.4)	168	(0.7)
Other/Undetermined	206	(2.2)	275	(4.0)	247	(4.3)	760	(3.4)
Total	9,571	(100.0)	6,928	(100.0)	5,710	(100.0)	22,478	(100.0)
Cumulative Incidence per 100,000***	183.1		901.1		1,028.9		336.2	

*Included in the total are 149 cases whose race/ethnicity is American Indian, Alaskan Native, Asian, or Pacific Islander and 120 whose race/ethnicity is unknown.
**See Table 1.
***Incidence was calculated as the cumulative number of AIDS cases among males, age 15 years and older, since the beginning of the epidemic, divided by estimates for the male population, age 15 years and older, by race/ethnicity in 1985. (See text for details.)

TABLE X-4—AIDS Cases among Female Adults/Adolescents by Exposure Category and Race/Ethnicity, and Cumulative Incidence per 100,000 Females, Aged 15 Years and Older, in New York State

	White		Black		Hispanic		Total*	
Exposure Category	Number	(%)	Number	(%)	Number	(%)	Number	(%)
Intravenous (IV) Drug Use	368	(60.2)	1,141	(63.4)	633	(60.7)	2,155	(61.8)
Hemophilia/Coagulation Disorder	2	(0.3)	1	(0.1)	0	(0)	3	(0.1)
Heterosexual Contact	142	(23.2)	488	(27.1)	352	(33.7)	992	(28.4)
Sex with Person at Risk for AIDS	142	(23.2)	360	(20.0)	352	(33.7)	864	(24.8)
Born in Pattern II Country**	0	(0)	128	(7.1)	0	(0)	128	(3.7)
Receipt of Blood Transfusion, Blood Components, or Tissue	56	(9.2)	27	(1.5)	12	(1.2)	97	(2.8)
Other/Undetermined	43	(7.0)	143	(7.9)	46	(4.4)	242	(6.9)
Total	611	(100.0)	1,800	(100.0)	1,043	(100.0)	3,489	(100.0)
Cumulative Incidence per 100,000***	10.5		185.5		160.0		46.0	

*Included in the total are 13 cases whose race/ethnicity is American Indian, Alaskan Native, Asian, or Pacific Islander and 22 whose race/ethnicity is unknown.

**See Table 1.

***Incidence was calculated as the cumulative number of AIDS cases among females, age 15 years and older, since the beginning of the epidemic, divided by estimates for the female population, age 15 years and older, by race/ethnicity in 1985. (See text for details.)

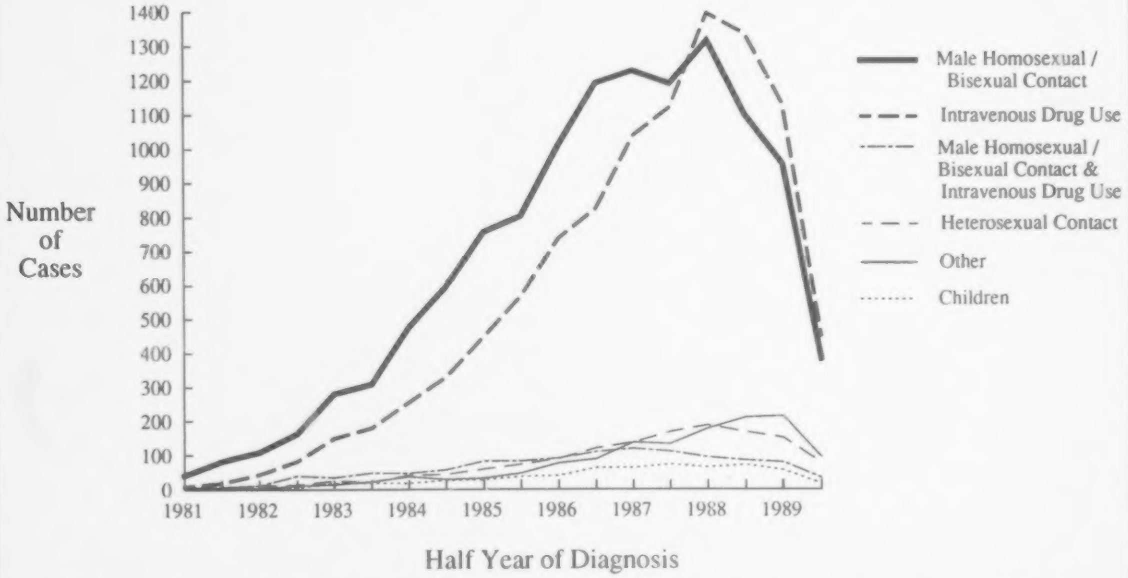


FIGURE 3—Number of AIDS cases among adult/adolescent exposure groups and among children by half-year of diagnosis, New York State

NOTE: Data for 1989 are incomplete due to the delay in case reporting.

The New York State DOH continues to monitor information from AIDS surveillance and the HIV seroprevalence studies to better address the needs of those already infected and to plan interventions that will limit the spread of HIV.

Summary

New York State has reported more AIDS cases (26,576) through November 1989 than any other state, accounting for 23 percent of all reported cases in the nation at the time. New York City has reported 22,231 (84 percent) of the statewide cases. The epidemiology of AIDS in New York State has been easily influenced by the large number of cases among IVDUs. Whereas intravenous drug use accounts for 16 percent of adult/adolescent cases in the rest of the nation,

TABLE X-5—Comparison of the Number and Incidence of Female AIDS Cases with HIV Seroprevalence among Women Giving Birth by Age Group, Race/Ethnicity, and Geographic Region in New York State 1988

Characteristics	Number of Cases and Incidence of AIDS per 100,000 Women in 1988 No.	Incidence*	Prevalence (per 100,000 Births) of HIV Seropositivity among Women Giving Birth in 1988** (N = 271,843)
Age Group (years)			
15–19	3	0.4	430
20–29	230	14.8	650
30–39	523	37.1	670
40–49	144	13.7	360
Race/Ethnicity			
White	145	4.0	120
Black	466	77.2	1,790
Hispanic	278	68.6	1,250
Other/Unknown	11	13.0	340
Geographic Region			
New York City	776	40.3	1,190
New York City Vicinity	80	7.9	260
Mid-Hudson Valley	13	5.8	220
Upstate Urban	24	3.5	130
Upstate Rural	7	0.8	80
Total	900	19.1	630

*Based on the number of AIDS cases among women ages 15–49 years and the 1985 population projections for women of this age group in New York State.

**Based on newborn HIV seroprevalence testing.

it accounts for 39 percent of the cases in New York State. Intravenous drug use is the leading exposure category among racial minorities, with a disproportionately high rate of cases among Blacks and Hispanics. The epidemic among women and children has also been severe, resulting from intravenous drug use by women, heterosexual transmission from drug users to women, and perinatal transmission to their children. ☐

Acknowledgments

The authors gratefully acknowledge the AIDS surveillance staff of the New York State and New York City Departments of Health for data collection, Rachel Stricof and Michael Davisson for newborn seroprevalence data, and Lloyd Novick for manuscript review. Surveillance activities were partially supported with funding through cooperative agreements with the Centers for Disease Control.

References

1. Barre-Sinoussi F, Chermann JC, Rey F, *et al:* Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). Science 1983; 220:868–871.
2. Gallo RC, Salahuddin SZ, Popovic M, *et al:* Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. Science 1984; 224:500–503.
3. Centers for Disease Control: Update on acquired immune deficiency syndrome (AIDS)—United States. MMWR 1982; 31:507–514.
4. Centers for Disease Control: Prevention of acquired immune deficiency syndrome (AIDS): Report of inter-agency recommendations. MMWR 1983; 32:101–103.
5. World Health Organization: Acquired immunodeficiency syndrome (AIDS): WHO/CDC case definition for AIDS. WHO Wkly Epidemiol Rec 1986; 61:69–72.
6. Centers for Disease Control: Revision of the CDC surveillance case definition for acquired immunodeficiency syndrome. MMWR 1987; 36(suppl no. 1S):3S–15S.
7. New York City Department of Health: AIDS trends to 1990. Morbidity and mortality. City Health Information 1990; 9:1–6.
8. Smith PF, Mikl J, Muse A, Duan S, Foster J: Assessment of the completeness of AIDS case reporting in New York State exclusive of New York City, using hospital discharge files. Abstract, 118th Annual Meeting of the American Public Health Association, New York, September 30–October 4, 1990.
9. Centers for Disease Control: HIV/AIDS Surveillance Report. Atlanta, GA: Centers for Disease Control, December 1989; 1–16.
10. Lifson AR, Hessol N, Rutherford G, *et al:* Natural history of HIV infection in a cohort of homosexual and bisexual men: Clinical and immunologic outcome, 1977–1990. Abstract ThC33, presented at the Sixth International Conference on AIDS, San Francisco, June 1990.
11. Bacchetti P, Moss AR: Incubation period of AIDS in San Francisco. Nature 1989; 338:251–253.
12. Munoz A, Wang M, Bass S, *et al:* Acquired immunodeficiency syndrome (AIDS)-free time after human immunodeficiency virus type 1 (HIV-1) seroconversion in homosexual men. Am J Epidemiol 1989; 130:530–539.
13. New York State Department of Commerce: Official population projections for New York State counties: 1980–2010. Albany, NY: New York State Department of Commerce, April 1985.
14. Novick LF, Glebatis DM, Stricof RL, MacCubbin PA, Lessner L, Berns DS: Newborn seroprevalence study: Methods and results. Am J Public Health 1991; 81(Suppl):15–21, Ch II.
15. New York State Department of Health: AIDS in New York State through 1988. Albany, NY: New York State Department of Health, 1989.

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American Journal of Public Health

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American Journal of Public Health

American Public Health Association

The American Journal of Public Health, published monthly, is the official Journal of the American Public Health Association, 1015 Fifteenth Street, NW, Washington, DC 20005; (202) 789-5600. APHA is a nongovernmental professional society founded in 1872, representing all disciplines and specialties in public health.

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Information on Manuscript Preparation is published in this Journal in January. The Journal publishes reports of original research, demonstrations, evaluations, and other articles covering current aspects of public health. Manuscripts are accepted for consideration with the understanding that they, or their essential substance, have not been published previously or accepted for publication and are not under consideration elsewhere.

Subscription Price is $80/year US; $120/year international. Single copies $10 US, $14 international (surface delivery), $22 international (air delivery). Prepaid only.

Postal Information: Second class postage paid at Washington, DC, and additional mailing offices. Postmaster: Send address changes to American Journal of Public Health, 1015 Fifteenth St., NW, Washington, DC 20005. AJPH allotted $40; TNH allotted $6 of APHA membership dues. Information on individual membership appears elsewhere in this Journal, or contact: Director, Membership Services, at APHA headquarters.

Advertising Sales: Mary Beth Kammann, Advertising Manager, APHA, 1015 Fifteenth St., NW, Washington, DC 20005; (202) 789-5668 or 5600. Advertising is accepted with the understanding that products and services are professionally related and meet the ethical standards of public health practice. Acceptance by the Journal does not indicate or imply endorsement by the Journal of APHA.

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The American Journal of Public Health is indexed in whole or in part in these data bases: Bibliographic Index of Health Education Periodicals, Biological Abstracts, Biology Digest, BRS Bibliographic Retrieval Service Inc, Chemical Abstracts, Cumulative Index to Nursing & Allied Health Literature, Environmental Information Center Inc., Environmental Periodicals Bibliography, Excerpta Medica, Grateful Med, Hospital Literature Index, Hospital Management Review, Human Resources Abstracts, Index Medicus (Abridged), MEDLARS, MEDSOC, Nuclear Science Abstracts, Physical Education Index, Public Affairs Information Service, Public Health Reviews, Safety Science Abstracts, Sage Family Studies Abstracts, Science Citation Index, and Social Work Research and Abstracts.

rence in the community . . .[1] of a group of illnesses of similar nature, clearly in excess of normal expectation." The attribution of this particular epidemic to the adverse effect of postaccident stress provides new and potentially important insights into the etiology of cancer and into the circumstances that may eventually need to be managed to better protect the public health. The idea that stress may be an environmental culprit could change many aspects of the way we handle acute environmental problems like the Three Mile Island accident. The way such environmental events are publicized and managed may need to be reconsidered if further evidence supports the conclusion that stress can cause or promote a disease like cancer. Studies of cancer incidence at the Love Canal before the problem was publicized in 1978 provided no clear evidence of increased cancer risks even though chronic exposure to environmental toxins had taken place over years and decades.[3] However, since the entire neighborhood surrounding the Love Canal was relocated at the time the problem of toxic exposure was publicized in 1978, it has not been possible to determine whether the stress associated with publicity surrounding that event might have lead to increased cancer rates in the years after 1978.

It will be important for public health officials to devise expanded research strategies for past and future environmental episodes that consider every plausible hypothesis for disease etiology. Latency periods prior to clinically apparent cancer may be very long. The promotion of already initiated cancer cells, through a stress-induced neuroendocrine mechanism, is not a far-fetched idea. Parenthetically, we should remember that only three or four decades ago many doubted that tobacco smoke could cause lung cancer and that the environmental pollution during an intense London fog in 1952 could have been responsible for some 4,000 deaths. As we consider the plausibility of the stress explanation in the report by Hatch et al., it is important to observe that a temporary promotion of cancer should lead to a short period of increased risk, followed by a compensatory period of decreased risk. The decreased risk occurs because new cancers are not initiated by a promotional event. Promotion can only hasten the progression of an existing cancer to the stage when the disease becomes clinically apparent. Perhaps mortality rates for cancer will need to be added to such studies.

The theories of initiation and progression of cancer are complex, but in general, a promotional event will not produce new cancers, although it may appear to do so in the short run. The data presented by the authors are consistent with such a pattern. As the authors clearly recognize, it is also difficult to rule out the possibility that increased clinical surveillance, perhaps stimulated by the same anxiety that produced the stress, could produce a short-term increase followed by a compensatory decrease in cancer cases diagnosed among the study population. The fact that the initial increase did not occur until three years after the nuclear accident lends more support to the promotion hypothesis than the increase-in-clinical-surveillance hypothesis as the explanation for this phenomenon. Whatever the final explanation for this cancer cluster following the Three Mile Island nuclear accident, the cluster underscores the need for more epidemiologically driven studies of major and minor environmental accidents. The investigators who conduct such studies should keep an open mind to the potential etiological mechanisms by which the circumstances around us may cause human diseases, including cancer. ☐

Dwight T. Janerich, DDS, MPH

References

1. Hatch MC, Wallenstein S, Beyea J, Nieves JW, Susser, M. Cancer rates after the Three Mile Island nuclear accident and proximity of residence to the plant. Am J Public Health 1991; 81:719–724.
2. American Public Health Association. The Control of Communicable Diseases in Man (11th ed.). New York: American Public Health Association, 1970. p. 289.
3. Janerich DT, Burnett WS, Feck G, Hoff M, Nasca P, Polednak AP, Greenwald P, Vianna N. Cancer Incidence in the Love Canal Area. Science 1981; 212(June 19):1404–1407.

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Erratum

In: Kahn HS, Williamson DF, Stevens JA: Race and weight change in US women: the roles of socioeconomic and marital status. Am J Public Health 1991;81:319–323.

The abstract was improperly copy edited by staff, resulting in the deletion of some important information in the Results section. The Results section of the abstract should read as follows:

"After multiple adjustments, Black race, education below college level, and becoming married during the follow-up interval were each independently associated with an increased mean weight change. Using multivariate logistic analyses, Black race was not independently associated with an increased risk of major weight gain (change $\geq +13$ kg), but it was associated with a reduced likelihood of major weight loss (change ≤ -7 kg) (odds ratio = 0.64 [95% CI = 0.41, 0.97]). Very low family income was independently associated with the likelihood of both major weight gain (OR = 1.71 [95% CI = 1.15, 2.55]) and major weight loss (OR = 1.86 [95% CI = 1.18, 2.95])."

The Journal regrets the error.

Asthmatic Responses to Airborne Acid Aerosols

Bart D. Ostro, PhD, Michael J. Lipsett, MD, JD, Matthew B. Wiener, PharmD, and John C. Selner, MD

ABSTRACT

Background: Controlled exposure studies suggest that asthmatics may be more sensitive to the respiratory effects of acidic aerosols than individuals without asthma. This study investigates whether acidic aerosols and other air pollutants are associated with respiratory symptoms in free-living asthmatics.

Methods: Daily concentrations of hydrogen ion (H^+), nitric acid, fine particulates, sulfates and nitrates were obtained during an intensive air monitoring effort in Denver, Colorado, in the winter of 1987–88. A panel of 207 asthmatics recorded respiratory symptoms, frequency of medication use, and related information in daily diaries. We used a multiple regression time-series model to analyze which air pollutants, if any, were associated with health outcomes reported by study participants.

Results: Airborne H^+ was found to be significantly associated with several indicators of asthma status, including moderate or severe cough and shortness of breath. Cough was also associated with fine particulates, and shortness of breath with sulfates. Incorporating the participants' time spent outside and exercise intensity into the daily measure of exposure strengthened the association between these pollutants and asthmatic symptoms. Nitric acid and nitrates were not significantly associated with any respiratory symptom analyzed.

Conclusions: In this population of asthmatics, several outdoor air pollutants, particularly airborne acidity, were associated with daily respiratory symptoms. (*Am J Public Health.* 1991;81:694–702)

Introduction

Exposure to acidic aerosols and sulfates has been associated with increased mortality and respiratory morbidity in a variety of settings.[1–5] Controlled exposure chamber studies suggest that individuals with asthma are more sensitive to the bronchoconstrictive effects of acidic sulfates than individuals without known pulmonary disease.[6–9] While the effects of exposure to ambient particulates (which may include acid aerosols) on asthmatic symptoms have been studied epidemiologically, previously published investigations have not included concurrent daily measurements of ambient airborne acidity.[4,5,10–12] Since the US Environmental Protection Agency has recently begun to consider whether to promulgate a national ambient air quality standard (NAAQS) for acid aerosols, it is important to assess the effects of relatively low levels of exposure on individuals in typical, free-living conditions. Thus, the need to extrapolate from studies of animals or of volunteers in controlled human clinical studies is obviated. In this study we report the results of an analysis of the association between ambient airborne acidity and daily respiratory symptoms recorded by a panel of asthmatics in Denver, Colorado during the winter of 1987–88.

Particulate air pollution has been associated with increased asthmatic symptoms and respiratory hospital admissions.[4,5,10–12] In an analysis of daily diaries of 454 asthmatics in the Los Angeles area, Whittemore and Korn[10] reported increased probabilities of attacks on days with high oxidant and total suspended particulate (TSP) pollution. Interpretation of this study is limited, however, by (among other things) the inadequacy of TSP as a measure of respirable particulate exposure and by significant covariation of TSP and ozone. Using a modification of the analytic model developed by Whittemore and Korn,[10,11] Perry, *et al,*[12] investigated the effects of fine (<2.5 microns in aerodynamic diameter) and coarse (>2.5 and <15 microns) particulates on respiratory symptoms, peak flows, and bronchodilator usage in 24 asthmatics in Denver in winter 1979. Fine nitrates were associated with increased symptoms and bronchodilator usage, while fine sulfates were associated with increased bronchodilator usage. The implications of this analysis are unclear because of the small sample size, the relatively low levels of particulate pollution, and the absence of acid aerosol measurements. In an ongoing study in Ontario, Canada, summer hospital admissions for asthma have been associated with exposure to ozone, sulfates, and sulfur dioxide.[4,5] These constituents of the "acid summer haze" were highly correlated; however, recent evidence[5] suggests that ozone may be less important than sulfates since there was a low correlation between respiratory admissions and ozone during one month of particularly high ozone concentrations. Again, acid aerosols were not measured.

The respiratory effects of airborne particulates on asthmatics and others have been attributed to the irritant, acidic frac-

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Address reprint requests to Bart D. Ostro, PhD, California Department of Health Services, 2151 Berkeley Way, Annex 11, Berkeley, CA 94704. Dr. Lipsett is also with the Department of Health Services at Berkeley; Dr. Wiener is with Pharmatech Inc., Englewood, CO. Dr. Selner is with the Allergy Respiratory Institute of Colorado, Denver, CO. This paper, submitted to the Journal October 24, 1989, was revised and accepted for publication August 13, 1990.

tion of this heterogeneous mixture of pollutants.[13–15] In controlled chamber studies of healthy individuals and asthmatics, acidic sulfates (H_2SO_4 and NH_4HSO_4) have been associated with bronchoconstriction and increased bronchial reactivity.[6–9] In a study involving short exposures (30 minutes at rest followed by 10 minutes of exercise), Koenig, *et al*,[6] reported significant changes in flow rates and pulmonary resistance in adolescent asthmatics exposed to 100 $\mu g/m^3$ H_2SO_4. Some studies showed effects only at higher levels of exposure which exceed typical urban peak sulfate levels,[7–9] and some showed no effects.[16] Nitrates and nitric acid have been studied less than acidic sulfates. Overall, the results of chamber studies suggest that asthmatics may be an appropriate "sensitive population" to study epidemiologically in relation to airborne acidity.

An intensive daily air monitoring program in the metropolitan Denver area was conducted from November 1987 to February 1988 to investigate the chemical composition and particle size distribution of the winter "brown cloud." The Integrated Environmental Management Program of Region VIII of the US Environmental Protection Agency (EPA) conducted a complementary air monitoring program from November 1987 to March 1988. The joint monitoring effort included twice-daily measurements of particulates (sulfates, nitrates, and all particles less than 2.5 microns in diameter or PM2.5), airborne acidity, and sulfur dioxide at two sites. Aerosol acidity was measured using annular denuder instrumentation[17] and included nitric acid vapor and nanoequivalents of hydrogen ion (H^+) in particulate form, which was not further speciated (into H_2SO_4 or NH_4HSO_4). Meteorologic data were collected by the State of Colorado and by Stapleton Airport in Denver. This period of intense air monitoring provided an occasion to investigate whether exposure to fine acidic particulates is associated with respiratory effects in asthmatic individuals. No previously published epidemiologic studies have correlated concurrently measured airborne acidity with asthmatic symptoms and behavior.

Data and Methods

Study Population

Study participants were recruited from patients attending the clinic of one co-author (JCS). Diagnosis of asthma was made in each case by history and signs of airway obstruction on physical examination, confirmed by spirometric demonstration of obstruction reversible with a β-agonist bronchodilator (>15 percent change in FEV_1). Asthmatic patients were identified by clinic staff and were recruited for participation either during an office visit or by telephone and postcard contact. Denver residents between ages 18 and 70 were eligible to participate in the study if they had asthma currently managed with medication. Individuals with any other chronic medical condition that would restrict their activity were excluded.

This panel of asthmatics was asked to record, on a daily basis for several months, information about symptoms, medication use, utilization of medical services, indoor exposures, and other variables described below. In the context of the air monitoring efforts noted previously, a panel study has several advantages over other investigative designs. First, it provides a large number of observations, increasing the degrees of freedom and the stability of any estimates. Second, problems of confounding, omitted variables, and exposure assessment, while always present, are substantially reduced since individuals serve as their own controls over time. Thus, the impact of factors that vary daily, such as air pollution, can be isolated while other factors are held constant. Third, the consideration of a given sample in one metropolitan location over time eliminates the potential for any intercity confounding.

TABLE 1—Characteristics of Patient Population Participating in the Denver Asthma Study

Characteristics	Total Study Population (n = 256)	Diary Participants (n = 207)
Mean age	44.3	45.6
% Male	34	34
Race (% White/Black/other)	94.6/2.3/3.1	96.5/1.5/2
Education (%)		
College	62	62
High school	37	37
Less than high school	1	1
Employment status (% employed outside home/homemaker/retired/other)	71/10/15/4	70/10.5/13.5/6
Mean subjective asthma severity rating (0 = none, 4 = incapacitating)	1.78	1.74
% Daily Theophylline	56.4	58.0
% Daily oral steroids	21.6	18.9
% Current smokers	3.1	3.0

Health Measurements and Covariates

After giving informed consent, participants were required to fill out an intake questionnaire providing background data on demographics (age, sex, race, level of education, employment status, residential history), asthma severity and characteristic triggers and symptoms, medical history including medication use, smoking history, and previous environmental exposures. A diary instrument was designed to provide daily information on asthma symptoms, including the presence and severity on a scale of 0 to 4 (0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = incapacitating) of cough, wheeze, shortness of breath, chest tightness, and sputum production, as well as physician and emergency room visits. In addition, information was obtained on frequency of medication use, time spent outdoors, levels of exercise intensity and whether exercise occurred indoors or outside, indoor exposure to respiratory irritants (such as gas stoves, fireplaces, and environmental tobacco smoke), and occupational exposures. Participants were told that this was an investigation of environmental factors affecting asthma, but not that the principal variables of interest were air pollutants.

There were 330 intake questionnaires distributed (93 during office visits and 237 by mail) from November 15 to December 16, 1987. Of the initial group recruited, 256 returned the intake questionnaires. Study subjects were contacted by clinic staff intermittently throughout the study period (December 1987 through February 1988) to enhance compliance and continued participation. Two hundred and seven patients returned their daily diaries. There were similar distributions of demographic variables and asthma severity in the initial study population and those who returned the daily diaries (Table 1). The group un-

TABLE 2—Descriptive Statistics of Pollutant, Meteorological, and Health Variables

	N	Mean	Std Dev	Minimum	Maximum
Sulfates ($\mu g/m^3$)	74	2.11	2.47	0.12	12.65
Nitrates ($\mu g/m^3$)	54	6.83	17.56	0.01	24.5
PM2.5 ($\mu g/m^3$)	47	21.97	20.40	0.51	73.08
Sulfur dioxide ($\mu g/m^3$)	78	14.14	12.82	0.80	59.8
Nitric Acid ($\mu g/m^3$)	64	1.81	2.25	0.06	13.54
H^+ (neq/m^3)	27	10.07	9.52	2.00	41.31
SH+ (neq/m^3)	74	8.15	8.29	0.59	44.25
Minimum temp. (F)	105	17.71	11.97	−11.00	39.00
Asthma	105	16.33	3.84	7.41	30.56
Cough	105	15.06	4.26	7.41	32.00

SH^+ = Hydrogen ion readings including substitutions for missing values.

Asthma = Group probability (×100) of reporting a moderate or worse overall asthma condition on each day.

Cough = Group probability (×100) of reporting a moderate or worse cough on each day.

All pollutants are measured as the average from 9:00 am to 4:00 pm

TABLE 3—Correlation Coefficients for Pollution and Meteorological Variables

	SULF	SO_2	NITR	PM2.5	HNO_3	H^+	SH^+	Mintemp
SO_2	.51							
NITR	.20	.16						
PM2.5	.88	.64	.40					
HNO_3	.22	.37	.78	.68				
H^+	.66	.35	.40	NA	−.11			
SH^+	.86	.44	.15	.56	.23	1.0		
MINTEMP	−.45	.26	−.08	−.34	−.07	−.57	−.43	
RH	.35	.12	.15	.40	.17	.32	.34	−.34

SULF = Sulfates

SO_2 = Sulfur dioxide

NITR = Nitrates

PM2.5 = Particulate matter less than 2.5 microns in diameter

HNO_3 = Nitric acid

H^+ = Hydrogen ion

SH^+ = Hydrogen ion with substitution for missing values (see text)

Mintemp = Minimum daily temperature

RH = Relative humidity

NA = Not available

der study was predominantly White, female, employed, and well educated, with an average age of approximately 46. The mean subjective asthma rating of moderate to severe is supported by the relatively large proportion of individuals taking daily oral theophylline and steroid preparations.

Exposure Measurement

The ambient air pollutants incorporated in the analysis were daily measures of sulfates, nitrates, PM2.5, nitric acid, hydrogen ion (H^+), and sulfur dioxide. Two different pollutant averaging times were considered initially: the 24-hour average (ending at 4:00 pm on the reporting day) of pollutant readings from the monitor in downtown Denver, located about two miles from the clinic; and the daytime average (9:00 am to 4:00 pm) from the same monitor. Since these averaging times were highly correlated (usually r > .90), only the daytime average was used in subsequent analysis. A second monitor, located about seven miles away in the northwestern suburb of Arvada, also provided measurements of these pollutants. Table 2 provides descriptive statistics for the pollutant, meteorologic and health variables. We found no statistically significant relationship between any pollutant and either the number of the day of the survey period or whether the reading was on a weekday or weekend and no significant time trend for either daily minimum temperature or daily humidity.

There was substantial covariation among some of the pollutants; the highest correlations were observed between PM2.5 and sulfates (r = .88), and between nitrates and nitric acid (r = .78). Sulfates and H^+ were both negatively correlated with maximum daily temperature (Table 3). Of the pollutants measured on a daily basis, sulfates had the most complete data. Values for missing days of PM2.5 and nitrates were derived from stepwise regressions using all other pollutants. PM2.5 could be predicted well by concurrent values of sulfates and nitrates (r = .97), and nitrates could be predicted by nighttime (4:00 pm to 9:00 am the following day) PM2.5 readings (r = .86).

The data on H^+ required additional attention. Because of problems in sample processing, the H^+ data were available only for half of the study period. The first seven days of H^+ readings were extremely high, with a mean of 288 nanoequivalents per cubic meter (neq/m^3), versus a mean of 10.1 for the following 28 days. The hydrogen ion concentrations during the first seven days were also unrelated to any other pollutant or meteorologic variables, while during the next 28 days there was a correlation, as expected, between H^+ and sulfates (r = .66). Consequently, the first seven days were dropped from the subsequent analysis. For H^+, missing values for the entire survey period were predicted using regression results with concurrent sulfate as the explanatory variable. With these substitutions, the daytime mean of H^+ was 8.15 neq/m^3 or approximately 0.4 $\mu g/m^3$ measured as sulfuric acid, with the highest daily average of 44 neq/m^3. All subsequent analysis for H^+ combined actual and imputed values. The one-hour peak may be three to four times higher than this longer-term average. These levels are typical of urban areas.[18] Analysis of the two monitors recording H^+ indicated that the concentrations of airborne acidity were fairly evenly distributed throughout the Denver area; the daily correlation of the readings was 0.88. Thus, the one downtown monitor was used to represent daily levels of H^+. Ozone concentrations, which frequently are highly correlated with sulfates and acids in the summer, were essentially at background levels through the winter in Denver (i.e. the maximum one-hour concentration was 0.042 ppm) and do not correlate with H^+ concentrations.

Previous research[19,20] suggests that incorporating information on exercise and time spent outdoors will improve the estimate of exposure. Therefore, we developed a measure of exposure that involved the product of the ambient pollution level, the duration of exposure, the time spent indoors and outdoors, and the ventilation rates based on exercise level (i.e. ventilation rates of 7, 10, 25, and 40 liters per minute for resting, mild, moderate, or strenuous exercise, respectively). Since the actual time and duration of exercise was not recorded in the diary, it was assumed to occur during the day and to last for one hour. Hours outdoors and exercise levels were taken directly from the daily diary, while the penetration ratio of indoor to outdoor levels of H^+ of 0.3 was derived from the published literature.[21] This adjustment for exposure was crude but represents an improvement over simply using outdoor levels of air pollution. The extent of the reduction in measurement error (and improvement in exposure assessment) is specifically tested in the subsequent analysis.

All data from questionnaires, daily diaries, air quality, and meteorologic monitoring were coded and edited in a SAS format for analysis.

TABLE 4—Regression Coefficients Relating Ambient Air Pollution and Exposure-adjusted Air Pollution to Moderate Asthma Symptoms, Corrected for Autocorrelation (standard error in parentheses)

Independent Variables	Cough	Asthma Rating	Cough	Asthma Rating
Constant	5.9	0.026	−0.15	−7.9
Symptom on previous day	74.8^a	83.0^a	76.6^a	97.1^a
	(14.5)	(8.1)	(15.6)	(8.3)
Log (H^+)	0.76^b	0.31		
	(0.38)	(0.42)	—	—
Exposure-adjusted log (H^+)	—	—	1.09^b	1.00^c
			(0.51)	(0.57)
Day of Survey	−0.067^c	0.005	−0.76^b	0.014
	(0.038)	(0.035)	(0.041)	(0.036)
Weekend	−0.85^c	−0.04	−1.5^b	−0.39
	(0.66)	(0.79)	(0.71)	(0.82)
Gas stove	6.53^a	6.97^a	8.25^a	6.69^a
	(1.75)	(1.88)	(1.94)	(2.03)
Minimum temperature	0.012	0.003	0.055	0.029
	(0.039)	(0.044)	(0.044)	(0.050)
R^2	.44	.37	.46	.37
# of observations	7417	7420	6364	6365
(# of people)	(168)	(168)	(163)	(165)

NOTE: a) significant at $p < .01$; b) significant at $p < .05$; c) significant at $p < .10$. All coefficients are ×100.

Statistical Methods

For this analysis, two different health outcomes were used as dependent variables in the multiple regressions: the probability of a respondent reporting on a given day a moderate (or worse) cough and the probability of reporting a moderate (or worse) rating of overall asthma status. The daily diary question regarding asthma status follows queries about the severity of wheezing, shortness of breath, chest tightness, and sputum production and should, therefore, reflect the respondents' subjective overall ratings of their individual asthma symptoms. As a daily average, 16.33 percent of the respondents (range 7.4 to 30.6 percent) reported a moderate or worse asthma day and 15.1 percent (range of 7.4 to 32 percent) reported a moderate or worse cough (see Table 2).

We estimated effects on each health outcome using ordinary least squares regression models. Variables included in each regression were minimum daily temperature, and binary variables indicating daily use of a gas stove and if the response occurred on a weekend. Since reporting of symptoms decreased during the course of the study, we also included the time since the start of the study. The probability of a symptom on the previous day was included as an explanatory variable in light of previous reports.[10,11] Daily relative humidity was examined and eliminated from the model after it was consistently found to be unrelated to any of the health outcomes, as were several time-invariant variables such as sex and education. Each pollutant's effect on health was estimated by entering it separately into the regression equation.

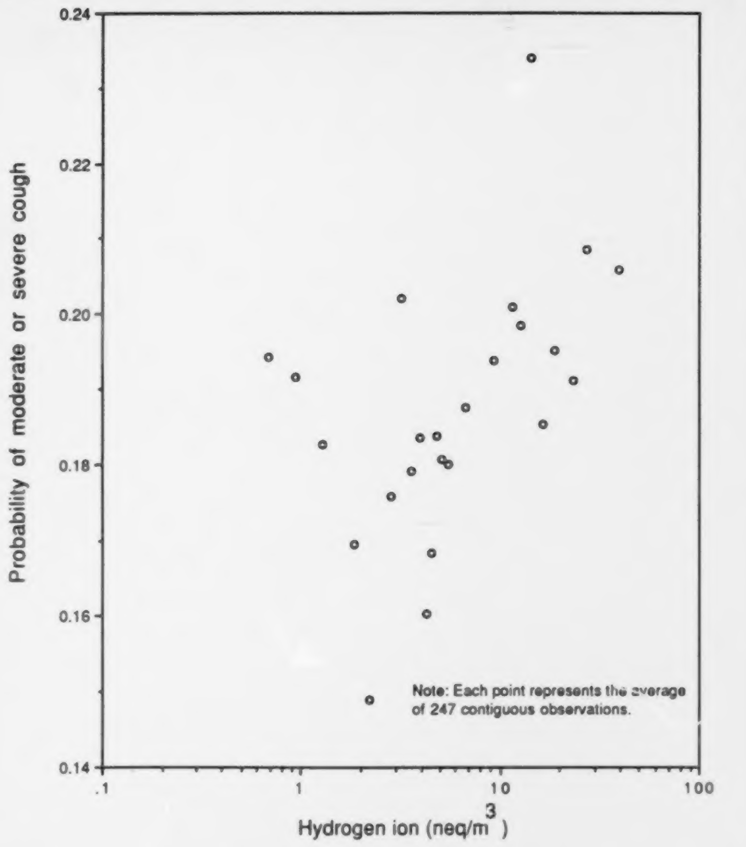
We also fitted a linear model of outcome using the logarithm of the pollutant, and a logistic model. There was little difference in model fit across the functional forms, but the log value of pollution provided the greatest association with the health outcomes. Thus, ordinary least squares with a log pollution term was used throughout the analysis. The similarity of results between ordinary least squares and logistic models probably was due to the relatively high rates of symptom incidence.

Significant autocorrelation was present, leading to biased estimates of the standard error of the estimated coefficients, and incorrect tests of significance.[22] The standard correction for autocorrelation involves estimation of a parameter (ρ) relating the regression residual at time t, r_t, to the residual for the previous time interval, r_{t-1}. When this feature is added to the estimation, the new model will have adjusted residuals which are uncorrelated. However, in this case, the procedure is biased and inconsistent because a lagged dependent variable (probability of symptom of the previous day) is included as an explanatory variable, and is correlated with the error term. Thus, the two-step procedure suggested by Fuller[23] was used. First, a predictive model was developed for the lagged value of the health effect (y_{t-1}) by regressing it on several independent variables, including time since start of the survey, specific air pollutants, minimum daily temperature, and humidity. Then the predicted value of y_{t-1} was used as an instrumental variable along with the other explanatory variables in the regression equation for y_t. Since the predicted value of the lagged dependent variable is uncorrelated with the adjusted residuals, the estimates are consistent. The estimation was performed using Proc Autoreg in SAS[24] with a maximum likelihood procedure that estimates both the regression coefficient and the autoregressive term.

Additional sensitivity analysis was conducted on the data, including a test for the influence of extreme observations, examination of other respiratory symptoms, and utilization of the exposure-adjusted measure of air pollution described above.

Results

Table 4 provides the ordinary least squares regression results for H^+, with the correction for autocorrelation. In the first two models, the dependent variables are the probability (×100) of reporting moderate or severe cough, and the probability of a moderate or worse asthma rating, respectively. The results indicate a statistically significant relationship between H^+ concentration and cough ($p < .01$) but not with overall asthma rating. Approximately 40 percent of the variation in the



Note: Each point represents the average of 247 contiguous observations.

FIGURE 1—Association of Moderate or Severe Cough with Hydrogen Ion

probability of reporting these symptoms was explained by the regression. For both cough and asthma status, reporting that symptom on the previous day was significantly related ($p < .0001$) with reporting a moderate or worse symptom. For cough, the day of the survey, responding on a weekend, and using a gas stove were statistically significant. Using overall asthma rating as the dependent variable, the only other significant risk factor (besides symptom on previous day) was usage of a gas stove ($p < .001$). For both cough and asthma status, autocorrelation corrections were appropriate and a second-order autocorrelation model was indicated. Figure 1 displays a plot of the relationship between hydrogen ion and moderate or severe cough.

Next, models with exposure-adjusted measure of H^+ were examined (Table 4). These models adjust outdoor air pollution levels for daily exercise levels and time spent outdoors. The results indicate that the exposure-adjusted measure of H^+ had a statistically significant association with both moderate cough ($p < .05$) and general asthma rating ($p < .10$). Figures 2 and 3 display these relationships.

Additional models correcting for autocorrelation were examined for each of the other pollutants, including sufates, sulfur dioxide, PM2.5, PM2.5 with substitutions for missing values, nitrates, and nitric acid. Each pollutant was considered individually and regressed first on cough and then on asthma rating and on shortness of breath. The other explanatory variables used in the multiple regression were those used above. Thus, Table 5 gives the results of these 18 separate regressions, each with one pollutant included in the equation. For comparison, the previous results for H^+ and exposure-adjusted H^+ are also included in the table.

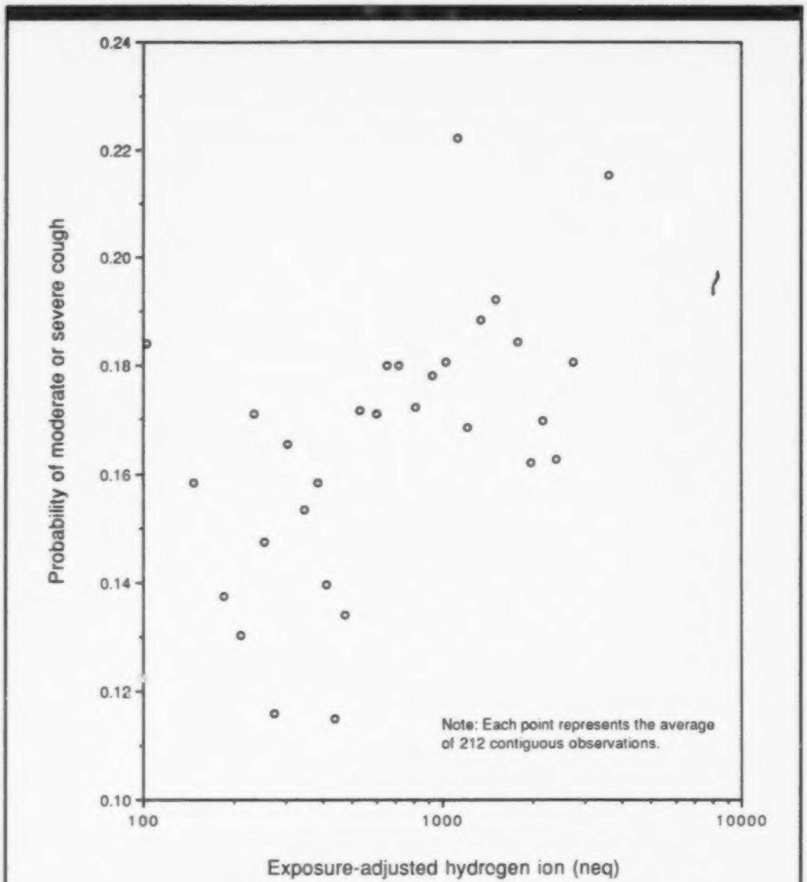
The results suggest that H^+, besides being associated with cough and overall subjective asthma rating, is also related to the reporting of moderate or worse shortness of breath. Besides H^+, the only other associations between individual air pollutants and these symptoms include: 1) sulfates and the probability of reporting moderate or worse shortness of breath ($p < .05$); and 2) PM2.5 and asthma rating ($p < .10$), after substitutions for missing values of PM2.5 were made, increasing the study duration from 46 to 75 days. Sulfur dioxide, nitrates, and nitric acid were not associated with moderate cough, asthma, or shortness of breath. The results for pollutants other than H^+ did not change when exposure-adjusted measures were used.

Additional analyses of the relationship of H^+ and asthmatic symptoms were conducted. The sensitivity of the models to the influence of extreme observations was tested using statistics proposed by Belsley, *et al.*[25] The test indicated that only the data from day = 2 had a relatively large influence on the regression results. The autoregressive model was rerun with that observation deleted, resulting in estimated regression coefficients that were essentially unchanged. Next, one- and two-day lags in H^+ were examined, indicating that contemporaneous measures of pollution provided the best association with asthma status. Finally, several time-invariant factors including education, sex, and whether an individual was recruited through office visit or by telephone were individually examined in the regression model. Their inclusion did not alter the estimated pollution effects.

Discussion

This analysis tested for an association between respiratory symptoms and several different air pollutants in a group of generally moderate to severe adult asthmatics. The results indicate a statistically significant association between ambient concentrations of hydrogen ion and the proportion of the subjects reporting moderate or worse cough and shortness of breath, with a lesser association observed in relation to the study subjects' overall asthma rating. Several lines of statistical and biomedical evidence implicate hydrogen ion as the principal pollutant of concern. First, although hydrogen ion was moderately correlated with minimum temperature and humidity (see Table 3), neither of these potentially confounding meteorologic variables was associated with the respiratory effects under investigation. Thus, it is unlikely that the estimated effects of hydrogen ion should be attributed to meteorologic changes over the survey period.

Second, confounding due to exposure to ambient ozone, which may cause



Note: Each point represents the average of 212 contiguous observations.

FIGURE 2—Association of Moderate or Severe Cough with Exposure-Adjusted Hydrogen Ion

exacerbations of asthma, was not a factor here. In studies linking health effects in asthmatics with resulting from summer haze in Southern Ontario[4] and particulate matter in Los Angeles,[10] ozone was highly correlated with the particle measure used in each study (sulfates and total suspended particulates, respectively), which in turn could be a surrogate for acid aerosol. In the Denver winter of 1987–88, ozone was generally found at background levels, as expected, with a maximum hourly concentration of 0.042 ppm.

Third, sensitivity analysis indicates that the findings were robust to functional form, specification, and influential observations. Linear and logistic functional forms provided similar results, and the estimated effect of air pollution was not affected by the exclusion of influential observations. In addition, the regressions were corrected for autocorrelation. Therefore, the results of the above analysis indicate that the symptoms reported are not likely to be due to a statistical anomaly.

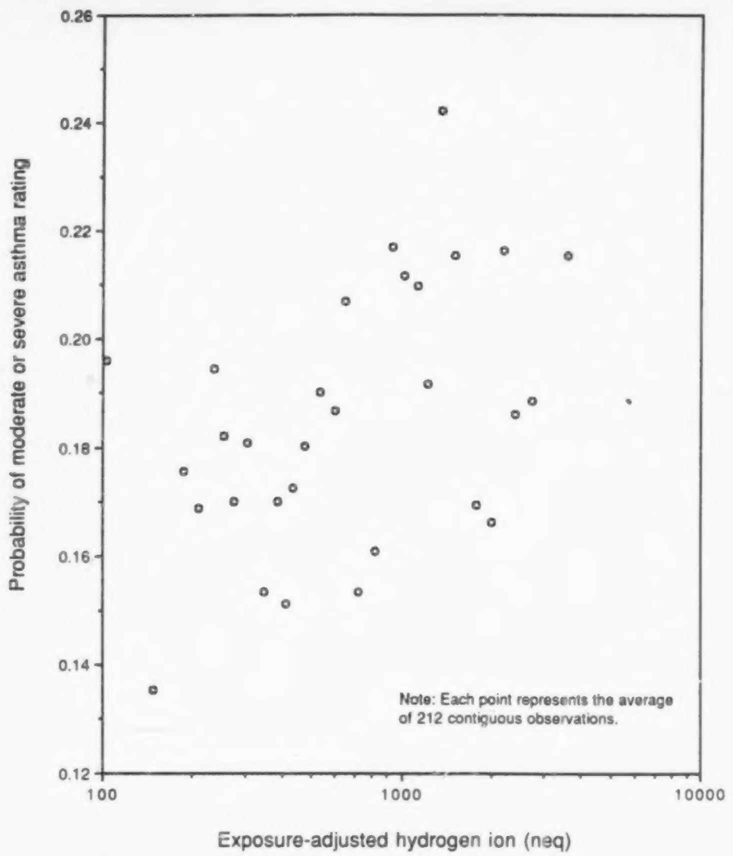
The estimated impact of air pollution is greater when an adjustment for actual exposure is undertaken. For example, as indicated in Table 4, after adjusting exposure for intensity and location of exercise, time spent outdoors, and penetration rate of H^+, the estimated effect of air pollution on cough is 43 percent greater than that obtained from the unadjusted pollution measurements. This suggests that dose-response estimates for H^+ that do not incorporate behavioral factors affecting actual exposures may seriously underestimate the impact of air pollution.

Evidence from controlled exposure studies supports an etiologic role of hydrogen ion exposure in evoking asthmatic symptoms. Increasing sulfate aerosol acidity enhances the bronchoconstrictive effects in asthmatics in rough proportion to the acidity.[7] Exposure to 75 $\mu g/m^3$ of the nonacidic respiratory irritant, ferric sulfate, did not cause adverse effects on pulmonary mechanics or respiratory symptoms in normal or asthmatic subjects.[26] More recently, Fine, *et al*,[27] showed that buffered acid aerosols were significantly more potent bronchoconstrictors than unbuffered acids at the same pH, suggesting that the total available hydrogen ion is a critical determinant of airway responsiveness. The relationship between asthmatic symptoms and pulmonary function changes due to acid exposure is not clear-cut; however, some studies indicate that symptoms (especially cough) precede accompanying changes in respiratory mechanics, although this has not been consistently observed.[27–29]

This study suggests that hydrogen ion is the pollutant of primary concern. Although both sulfates and hydrogen ion were significantly related to the probability of reporting moderate or worse shortness of breath, only exposure-adjusted hydrogen ion was associated with the probability of reporting both a moderate or worse cough and asthma rating. Concentrations of particulate matter less than 2.5 microns were mildly associated with overall asthma ratings, but not with either cough or shortness of breath. Supporting evidence comes from a study of both asthmatics and healthy individuals,[30] in which ambient sulfates were not associated with acute respiratory symptoms, but airborne pH levels were. The importance of hydrogen ion over total sulfates or particle mass has also been suggested by the US EPA.[31] Since sulfate and particle measurements may include both acidic and nonacidic exposures, they incorporate measurement error which would bias downward the estimated regression coefficients and level of significance.

Of additional interest is the lack of association between nitrates or nitric acid and respiratory symptoms. Limited data from controlled chamber studies suggest that brief (10–16 min) exposure to high concentrations (up to 1,000 $\mu g/m^3$) of inorganic nitrate ($NaNo_3$) aerosols do not evoke respiratory symptoms or changes in pulmonary function in mild asthmatics.[32,33] Thus, there is some (admittedly sparse) clinical evidence to support the notion that particulate nitrates *per se* would not be expected to cause the effects reported here.

Our findings ostensibly conflict with those of Perry, *et al*,[12] who reported a mild association between respiratory symptoms and nitrates, but not sulfates, in a sample of 24 asthmatics. There may be at least three reasons for this discrepancy. First, the small sample size limits the generalizability of their findings. Second, the



Note: Each point represents the average of 212 contiguous observations.

FIGURE 3—Association of Moderate or Severe Asthma Rating with Exposure-Adjusted Hydrogen Ion

rate of respiratory symptom reporting in that investigation was very low relative to ours. Third, there is a difference in pollutants under consideration and their dispersion patterns. Perry, *et al*, measured nitrates, sulfates, sulfur dioxide, and PM2.5 but not acid aerosols. In the Perry study and the present study the correlations among sulfates, sulfur dioxide, PM2.5, and temperature were remarkably similar, but nitrates were less correlated with other pollutants in the present study. This suggests that the sources of nitrates may have been quite different. Prior to the use of nylon back-up filters in the mid-1980s, nitrates were commonly measured inaccurately. Population growth and changes in automobile travel patterns also may have led to a change in exposures. In addition, during the winter of 1987–88, the major electric utilities, as part of a clean air campaign, burned natural gas instead of coal for alternate 15-day periods. These factors may have altered the dispersion pattern of nitrates, relative to sulfates and other pollutants, and the subsequent exposures of residents of the metropolitan area.

Since only about half of the individuals in this study completed their diaries every day, we investigated the potential for bias due to variable daily response rates. We found no relationship between air pollution and the number of people responding on a given day. We also investigated whether health status influenced response rates and found no relationship between overall severity of asthma and the number of days that people filled out the daily diaries. We infer that responses recorded in the daily diaries represent those of the group as a whole.

In this study population, the products of gas consumption appeared to exacerbate asthmatic symptoms. Peak indoor exposures to nitrogen dioxide (NO_2) resulting from gas stove use have been reported to range from 0.2 to 1.7 ppm,[34] which includes concentrations at which effects on symptoms or bronchial reactivity in asthmatics have been reported.[35,36] The concentrations of NO_2 and other constituents of natural gas combustion were not measured, but it is plausible that some symptoms were attributable to such indoor exposures.[37]

The results of this study, while supporting an association between asthmatic symptoms and ambient hydrogen ion exposure, should be interpreted with caution since pollutant measurements from fixed-site monitors cannot accurately represent individual exposures. Further, asthmatics may change their activity or medication use in order to reduce or eliminate symptoms related to air pollution; this would reduce the estimated effect of air pollution. Adjusting the pollution exposures for exercise and time outdoors increased the magnitude and precision of the estimate.

This study has several strengths, including the prospective nature of the investigation, the relatively large sample size, and the screening of the survey participants. Unlike previous large-scale studies of asthmatics, the sample for this study was recruited from one private practice at one location. Medical and personal histories were fully documented. Since the study was conducted as a time-series in one city over a relatively short period of time, geographic confounding factors were minimized, as was potential confounding due to ozone exposure. Relevant airborne acidity data, never previously collected concurrently with asthmatic symptom data, were also available.

This study provides evidence from a free-living population that is qualitatively consistent with the results of several controlled clinical investigations. Furthermore, our analysis indicates that, among moderate to severe asthmatics, respiratory symptoms occur at average hydrogen ion concentrations substantially lower than those at which effects on pulmonary mechanics have been observed in the laboratory.[7,9,28,29] That we were able to detect acute symptoms at the ambient concentrations measured was somewhat surprising. However, the study population was in general more severely afflicted than asthmatics volunteering for chamber studies. Some patients with severe asthma are known to have airway mucus with low pH, which may increase their susceptibility to acidic environmental insults.[38] Furthermore, the Denver winter temperatures and low relative humidity may have potentiated the effects of the airborne acidity (although we did not observe any obvious independent or interactive effects of these factors in the regression analysis). Cold dry air has been reported to enhance

TABLE 5—Estimated Regression Coefficients, Corrected for Autocorrelation, for Pollutants Examined Individually (standard error in parentheses)

Pollutants*	Asthma	Cough	Shortness of Breath
H^+	0.31	0.76[a]	1.05[a]
	(0.42)	(0.38)	(0.42)
Exposure adjusted H^+	1.00[b]	1.09[a]	1.33[a]
	(0.57)	(0.51)	(0.57)
Sulfate	0.24	0.42	0.77[a]
	(0.39)	(0.35)	(0.38)
SO_2	0.26	0.27	0.04
	(0.38)	(0.33)	(0.38)
PM2.5	0.06	0.12	−0.35
	(0.53)	(0.43)	(0.52)
PM2.5(Subs)	0.06[b]	0.14	0.29
	(0.03)	(0.29)	(0.38)
Nitrate	−0.25	−0.21	0.10
	(0.25)	(0.94)	(0.25)
Nitric acid	−0.03	−0.32	−0.10
	(0.35)	(0.34)	(0.34)

a) significant at $p < .05$; b) = $p < .10$

PM2.5(Subs) = substitute for missing values of PM2.5 = 8.67 • sulfate + 1.071 • nitrate

*All pollutants are in log terms. Besides the air pollutant, all equations include day of the survey, symptom on previous day, an indicator for weekend vs. weekday, use of gas stove, and minimum temperature as explanatory variables and are corrected for autocorrelation.

All coefficients are ×100.

bronchoconstriction caused by sulfur dioxide in one of the few experiments investigating interaction between physical and chemical exposures.[39] In other contexts, observational studies have found air pollution effects at concentrations below those at which chamber studies reported positive results.[40] This suggests that factors that are typically not included in chamber studies may play an important role in determining individuals' responses to air pollution. ☐

Acknowledgments

The authors would like to thank Steve Arnold of the Colorado Health Department, Mark Komp, Ken Lloyd, Steve Frey, and Robert Stevens of the US Environmental Protection Agency, and Suzy Rothschild of E. H. Pechan & Associates for assistance in collection, preparation, and quality control of air monitoring and health effects data. We would also like to thank Petros Koutrakis of the Harvard School of Public Health for supplying additional annular denuders.

An earlier version of this paper was presented at the 82nd Annual Meeting of the Air Pollution Control Association, June 1989.

References

1. Thurston GD, Ito K, Lippmann M, Hayes C: Reexamination of London, England, mortality in relation to exposure to acidic aerosols during 1963–1971 winters. Environ Health Perspect 1988; 79:73–82.
2. Ostro BD: A search for a threshold in the relationship of air pollution to mortality: A reanalysis of data on London winters. Environ Health Perspect 1984; 58:397–399.
3. Ware JH, Ferris BG, Dockery DW, Spengler JD, Stram DO, Speizer FE: Effects of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children. Am Rev Respir Dis 1986; 133:834–842.
4. Bates DV, Sizto R: Air pollution and hospital admissions in Southern Ontario: The acid summer haze effect. Environ Res 1987; 43:317–331.
5. Bates DV, Sizto R: The Ontario air pollution study: Identification of the causative agent. Environ Health Perspect 1989; 79:69–72.
6. Koenig JQ, Pierson WE, Horike M: The effect of inhaled sulfuric acid on pulmonary function in adolescent asthmatics. Am Rev Respir Dis 1983; 128:221–225.
7. Utell MJ, Morrow PE, Speers DM, Darling J, Hyde RW: Airway responses to sulfate and sulfuric acid aerosols in asthmatics. Am Rev Respir Dis 1983; 128:444–450.
8. Utell MJ, Morrow PE, Hyde RW: Airway reactivity to sulfate and sulfuric acid aerosols in normal and asthmatic subjects. J Air Pollut Control Assoc 1984; 34:931–935.
9. Utell MJ: Effects of inhaled acid aerosols on lung mechanics: Analysis of human exposure studies. Environ Health Perspect 1985; 63:39–44.
10. Whittemore AS, Korn EL: Asthma and air pollution in the Los Angeles area. Am J Public Health 1980; 70:687–696.
11. Korn EL, Whittemore AS: Methods for analyzing panel studies of acute health effects of air pollution. Biometrics 1979; 35:795–802.
12. Perry GB, Chai H, Dickey DW, *et al:* Effects of particulate air pollution on asthmatics. Am J Public Health 1983; 73:50–56.
13. Lippmann M: Airborne acidity: Estimates of exposure and human health effects. Environ Health Perspect 1985; 63:63–70.
14. Goldsmith JR, Friberg LT: Effects of air pollution on human health. *In:* Stern AC (ed): Air Pollution, 3rd Ed, vol. II. New York: Academic Press, 1977; 458–610.
15. Sun M: Acid aerosols called health hazard. Science 1988; 240:1727.
16. Sheppard D, Balmes JR, Christian D: Effects of temperature and particle size on acid aerosol-induced bronchoconstriction. Report to the California Air Resources Board; 1988.
17. Koutrakis P, Wolfson JM, Spengler JD: An improved method for measuring aerosol strong acidity: Results from a nine-month study in St. Louis, Missouri and Kingston, Tennessee. Atmos Environ 1988; 22:157–162.
18. Lipfert FW, Morris SC, Wyzga RE: Acid aerosols: The next criteria air pollutant? Environ Sci Technol 1989; 23:1316–1321.
19. Spengler JD, Keeler GJ, Koutrakis P, Ryan PB, Raizenne ME, Franklin CA: Exposures to acidic aerosols. Environ Health Perspect 1989; 79:43–51.
20. Hazucha MJ: Relationship between ozone exposure and pulmonary function changes. J Appl Physiol 1987; 62:1671–1680.
21. Brauer M, Koutrakis P, Spengler JD: Personal exposures to acidic aerosols and gases. Environ Sci Technol 1989; 23:1408–1412.
22. Rosner B, Munoz A, Tager I, Speizer F, Weiss S: The use of an autoregressive model for the analysis of longitudinal data in epidemiologic studies. Stat Med 1985; 4:457–467.
23. Fuller W: Introduction to Statistical Time Series. New York: John Wiley and Sons, 1976; 429–446.
24. 1984 SAS/ETS User's Guide, Version 5. Cary, NC: SAS Institute, 1984.
25. Belsley DA, Kuh E, Welsh RE: Regression Diagnostics. New York: John Wiley and Sons, 1980.
26. Kleinman MT, Linn WS, Bailey RM, Anderson KR, Whynot JD, Medway DA, Hackney JD: Human exposure to ferric sulfate aerosol: Effects on pulmonary function and respiratory symptoms. Am Ind Hyg Assoc J 1981; 42:298–304.
27. Fine JM, Gordon T, Thompson JE, Sheppard D: The role of titratable acidity in acid aerosol-induced bronchoconstriction. Am Rev Respir Dis 1987; 135:826–830.
28. Koenig JQ, Covert DS, Pierson WE: Effects of inhalation of acidic compounds on pulmonary function in allergic adolescent subjects. Environ Health Perspect 1989; 79:173–178.
29. Hackney JD, Linn WS, Avol EI: Acid fog: Effects on respiratory function and symptoms in healthy and asthmatic volunteers. Environ Health Perspect 1989; 79:159–162.
30. Zagraniski RT, Leaderer BP, Stolwijk JAJ: Ambient sulfates, photochemical oxidants, and acute adverse health effects: An epidemiological study. Environ Res 1979; 19:306–320.
31. US Environmental Protection Agency: Acid Aerosols Issue Paper. Washington, DC: EPA, Office of Health and Environmental Assessment, 1989.
32. Utell MJ, Swinburne AJ, Hyde RW, Speers DM, Gribb FR, Morrow PE: Air-

way reactivity to nitrates in normal and mild asthmatic subjects. J Appl Physiol 1979; 46:189–196.

33. Sackner MA, Dougherty RD, Chapman GA, Zarzecki S, Zarzemski L, Schreck R: Effects of sodium nitrate aerosol on cardiopulmonary function of dogs, sheep and man. Environ Res 1979; 18:421–436.
34. Goldstein IF, Andrews LR: Peak exposures to nitrogen dioxide and study design to detect their acute health effects. Environ Internat 1987; 13:285–291.
35. Kerr HD, Kulle TJ, McIlhany ML, Swedersky P: Effects of nitrogen dioxide on pulmonary function in human subjects. An environmental chamber study. Environ Res 1978; 19:392–404.
36. Mohsenin V: Airway responses to nitrogen dioxide in asthmatic subjects. J Toxicol Environ Health 1987; 22:371–380.
37. Lebowitz MD, Collins L, Holberg CJ: Time series analyses of respiratory responses to indoor and outdoor environmental phenomena. Environ Res 1987; 43:332–341.
38. Holma B: Effects of inhaled acids on airway mucus and its consequences for health. Environ Health Perspect 1989; 79:109–113.
39. Bethel RA, Sheppard D, Epstein J, Tam E, Nadel JA, Boushey HA: Interaction of sulfur dioxide and dry cold air in causing bronchoconstriction in asthmatic subjects. J Appl Physiol 1984; 57:419–423.
40. Spektor DM, Lippmann M, Thurston GD, *et al:* Effects of ambient ozone on respiratory function in healthy adults exercising outdoors. Am Rev Respir Dis 1988; 138:821–828.

Homeless Mentally Ill Will Benefit from Research Demonstration Projects

A unique collaborative effort to improve housing and services for homeless people who have a severe mental illness was announced recently by Health and Human Services Secretary Louis W. Sullivan, MD. The Department of Housing and Urban Development will join with HHS in supporting six research demonstration projects, each of which will combine housing assistance with mental health services.

The new projects will be carried out in five cities across the country and will be administered by the National Institute of Mental Health.

According to previous NIMH-sponsored research, one third of the homeless population has severe and persistent mental illnesses, including schizophrenia and mood disorders. Yet with adequate housing, treatment and support, most of these individuals can lead productive lives in the community.

NIMH funding for the new projects will be approximately $5.8 million in FY 1991 out of a planned total of $16.8 million over the next three years. HUD will contribute over $10 million in housing assistance. Projects will be located in Boston, Massachusetts; San Diego, California; Baltimore, Maryland; Cincinnati, Ohio; and two in New York City. In *Boston,* the project will evaluate two alternatives to housing homeless mentally ill individuals; consumer-managed group homes and independent living arrangements will be compared to determine which approach results in better housing stability with higher levels of functioning.

In *San Diego,* the research demonstration project will evaluate the effectiveness of linking comprehensive services to independent housing for severely mentally ill homeless persons.

In *Baltimore,* the project will test an "assertive community treatment" program designed to increase the clients' access to housing, as well as general health and mental health services.

The project in *Cincinnati* will compare the benefits of transitional housing and permanent housing.

In *New York City,* one research demonstration project will help individuals on the street obtain housing, such as single-room occupancy units, made available through a city/state agreement, while another project will be directed at mentally ill men who are preparing to leave a homeless shelter.

For further information, contact the Department of Health and Human Services, (301) 436-4536.

A Randomized Trial to Evaluate the Risk of Gastrointestinal Disease due to Consumption of Drinking Water Meeting Current Microbiological Standards

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ABSTRACT

Background: This project directly and empirically measured the level of gastrointestinal (GI) illness related to the consumption of tapwater prepared from sewage-contaminated surface waters and meeting current water quality criteria.

Methods: A randomized intervention trial was carried out; 299 eligible households were supplied with domestic water filters (reverse-osmosis) that eliminate microbial and chemical contaminants from their water, and 307 households were left with their usual tapwater without a filter. The GI symptomatology was evaluated by means of a family health diary maintained prospectively by all study families over a 15-month period.

Results: The estimated annual incidence of GI illness was 0.76 among tapwater drinkers compared with 0.50 among filtered water drinkers ($p < 0.01$). These findings were consistently observed in all population subgroups.

Conclusion: It is estimated that 35% of the reported GI illnesses among the tapwater drinkers were water-related and preventable. Our results raise questions about the adequacy of current standards of drinking water quality to prevent waterborne endemic gastrointestinal illness. (*Am J Public Health.* 1991;81:703–706)

Introduction

It has long been considered that water disinfected at 0.5 mg/L of free residual chlorine for 30 minutes at a pH less than 8, with a turbidity of less than 1 NTU would constitute minimal health risk to consumers.[1] Recently, however, increasingly sensitive analytic methods have permitted the detection of viruses[2] and parasites[3] in water meeting current water quality standards. This and several reported outbreaks of viral and parasitic diseases of waterborne origin[4] have prompted a review of the safety of current standards.[5] The suggested standards are based on the absence of fecal coliform bacteria in drinking water, fecal coliform bacteria being indicators of fecal pollution, and the ensuing risk of the presence of pathogenic microorganisms. The absence of total coliform bacteria in a majority of the samples collected is also recommended as an indicator of treatment efficiency.

While the current microbiological standards of water quality have virtually eliminated explosive outbreaks of waterborne disease,[6] there is no assurance that populations consuming drinking water prepared from sewage-contaminated wastes are free of less virulent gastrointestinal (GI) disease. While studies have addressed the health effects of recycled wastewaters[7,8] or of substandard drinking water,[9] all have relied on cases reported to the health care system to evaluate potential health effects. Other studies have addressed the health risks associated with chemicals in water or with disinfection byproducts.[10]

The present project was intended to directly and empirically answer the following question: Is there any measurable excess of GI illness related to the consumption of tap water prepared from sewage-contaminated surface waters and which meets current microbiological and physico-chemical water quality criteria?

Methods

Study Area

The study was carried out in a suburban area of Montreal comprised mainly of French Canadians with socioeconomic and education levels similar to the average of the Metropolitan Montreal area. The residents of this area are served by a single water treatment plant, which uses state-of-the-art treatment with pre-disinfection, flocculation by alum, rapid sand filtration, ozonation, and final disinfection by chlorine or chlorine dioxide. The raw water to be treated is drawn from a river which is mainly contaminated by human sewage discharges with little contamination from chemicals. The distribution system serves a population of about 40,000 people and distributes water meeting current microbiological and physico-chemical water quality standards.[5] A pilot study had shown that over 90 percent of the population drinks unmodified tap water and that the water was considered by the consumers to be of good to excellent quality.

Study Design

In order to determine the level of gastrointestinal illnesses attributable to drink-

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ing water, we carried out a randomized trial in which some households were left with their usual tap water and others were supplied with a domestic water filter which eliminates most chemicals and all particulate and microbiological matter from the tap water.

The sampling process was undertaken to enroll approximately 600 households for study, half randomly assigned to the filtered water group and half to the tap water group. Using a directory of inhabited addresses for the study area, households were selected at random and telephoned in random order. A screening questionnaire served to determine whether the household satisfied the five eligibility criteria: 1) owner-occupied; 2) French-speaking occupants; 3) regular consumers of tap water (as opposed to bottled or filtered water); 4) at least one child between the ages of 2 and 18 living in household; 5) willingness to participate in a longitudinal trial in which a random half of the households would have a filter installed. A total of 3,741 households were contacted before we achieved our quota of eligible study households. After randomization, there were 299 families in the filtered water group (1,206 individuals) and 308 families (1,201 individuals) in the tap water group.

The selection of eligible families was completed in January 1988 but the full-scale morbidity follow-up started in March 1988 after all filtration units had been installed in the appropriate households. Health outcomes of interest were monitored until June 1989. Because of the difficulty of monitoring during the summer vacation period there was a two-month gap in the surveillance during July and August 1988. We will refer to the March 1988 to June 1988 observation period as Period 1, and September 1988 to June 1989 as Period 2.

Intervention

For the tap water group participants, there was no intervention and no suggestion to modify their normal water consumption patterns.

For the participants allocated to the filtered water group, under-the-sink filtration units were installed by professional plumbers, at no expense to the family, during the start-up period of February-March 1988. The filters were commercial domestic reverse-osmosis units attached to the cold water line and placed under the kitchen sink. They were composed of a prefilter, an activated charcoal filter, a reverse osmosis membrane cartridge, and a 15-liter reservoir containing the purified water. During the reverse-osmosis process, tap-water is forced through a molecular filter which is permeable only to very small molecules such as water and some inorganic salts. Microorganisms, particulates, and most chemicals are rejected by the membrane and discharged to the drain. The filters were obtained, installed, and maintained by a local supplier. Prefilters and charcoal were changed after nine months of use or upon clogging. Efficiency of the units was monitored weekly by the family informant using a small conductivity tester. Water in the reservoir of such units is pressurized by the water pressure of the main inlet to the house and is available at all times in sufficient quantity for normal usage by the whole family.

Background Information and Health Outcomes

All participating households were initially visited in January and February 1988 by a nurse who carried out an interview on baseline factors and explained the way the health data would be collected. The baseline data included sociodemographic and medical history information on each household member. The health end-point of interest was GI symptomatology and this was determined primarily by means of a family health diary maintained prospectively by all study families for the whole period except for the two-month summer break. A simple self-administered questionnaire was designed to be completed in two-week blocks. One household member, usually the female head, was designated as the informant. The informant was responsible for noting for any household member on each calendar day each incident of the following events: nausea, vomiting, or diarrhea (soft or liquid). If any of these three symptoms was recorded, the presence of the following accompanying symptoms was to be noted: fever, cramps, muscular pain, cold or flu, sore throat, absence from work or school, visit to a doctor, or hospitalization. For any positive reports, we inquired as to possible reasons such as food overindulgence, pregnancy, and visits to countries with high endemic levels of GI illnesses. At the end of each two-week period, a nurse from our staff telephoned the informant and asked for the information recorded on the diary. The regular and frequent contact by our staff was intended to encourage reporting accuracy.

For the purpose of statistical analyses reported here, we defined an episode of highly credible GI (HCGI) symptoms as involving at least one of the following combinations: 1) vomiting or liquid diarrhea with or without confinement to bed, consultation with a doctor, or hospitalization, or 2) nausea or soft diarrhea combined with abdominal cramps with or without absence from school or work, confinement to bed, consultation with a doctor, or hospitalization. Episodes with plausible etiologies apart from the one under study (e.g. pregnancy) were excluded from consideration. An episode was further defined as consisting of one or more symptomatic days, with at least six consecutive symptom-free days between the episodes.

Statistical Analyses

Outcome parameters included the percentage of study subjects with episodes, the annual incidence of episodes and the duration of episodes. The study subjects can be seen as the family units or as the individuals within these family units.

Some analyses were crude and thus included no other covariates apart from group membership while others included covariates to adjust for some imbalance in the two groups. The covariates included in the model, in addition to tap water/filter water status were: age, sex, and geographic subregion. Estimates of incidence of HCGI illnesses in the two groups were derived by means of Poisson regression methods.[11] Since this technique counts each episode as an independent event, a correction had to be made for the correlation between repeat episodes by the same unit of observation. This was done using the "quasi-likelihood" approach,[12] which amounts to reducing the nominal significance of each result by a factor related to the amount of this correlation. Adjusted rates were estimated from the results of the fitted model. Each regression model gave an estimate of the p-value associated with the parameter for group membership. The preventable fraction of disease was computed by dividing the difference in rate between the tap water and filtered water by the rate in tap water group. The percentage of subjects with episodes over some period of time was estimated by two methods, logistic regression, and proportional hazards analysis. Both of these techniques are based on data relating to the first episode that the individual experienced during the period of observation; the number of episodes experienced is ignored. The logistic regression analysis[13] requires that the period of

TABLE 1—Distribution of Selected Individual Characteristics in the Two Study Groups at the Study Outset

	% in Group:	
Sub-groups	Filtered Water (N = 1206)	Tap Water (N = 1202)
Age group		
0–5	9.6	6.2
6–20	37.4	39.4
21–49	46.0	43.2
50+	7.0	11.2
Sex		
Female	49.4	49.5
Male	50.6	50.5

NOTE: There were some dropouts during the course of the study. The total sample sizes at the beginning of the second period were 1,194 in the filtered water group and 1,067 in the tap water group. The distributions of characteristics were almost identical in the second period to those shown above in the first period.

observation be equal for all study subjects. Thus this analysis had to be limited to subjects for whom there were no missing data during the entire field work period. However, this analysis provided estimates of percentages of study subjects in both study groups who had at least one episode. The proportional hazards analysis by contrast can use all study subjects irrespective of the length of observation period but it estimates a rate ratio rather than a pair of rates.[14]

Water Consumption

To evaluate whether subjects in the two study groups were in fact consuming the type of water we intended them to consume, we carried out a survey to estimate each person's water consumption habits during one week day and one weekend day in the preceding seven days. This questionnaire asked about water consumed inside and outside the home and listed different types of use (e.g. straight, in juice, in hot drinks). From this survey, we computed an estimate of weekly water consumption inside and outside the home for each person. The response rate to this supplemental self-administered questionnaire was 82 percent among tap water subjects and 84 percent among filter water subjects.

Microbiological Analysis of Water

Water from the filtration plant (raw and treated) from 25 sampling sites in the distribution system and filtration units was sampled at least weekly for bacteriological and physicochemical analysis. The bacteriologic parameters estimated were: heterotrophic plate counts at 20°C and 35°C, total coliforms and fecal coliforms; *Clostridium perfringens, Aeromonas hydrophila, Pseudomonas aeruginosa.* Large volume virological analysis of the raw and treated water from the plant were also performed at least weekly according to the methods described previously.[15] Physicochemical parameters evaluated were: pH, turbidity, and residual disinfectant concentration. All analyses were performed by the Montréal Laboratory of the Ministry of Environment of Québec according to *Standard Methods for the Examination of Water and Wastewater*[16] and to the Ministry recommendations.[17]

Results

Water Quality

River water used for the preparation of the drinking water was contaminated by fecal material of human origin as indicated by the high levels of the following pathogens and indicator microorganisms found in the raw water entering the plant: human enteric viruses (78 viruses/100L), total coliforms (57,530 cfu/L), fecal coliforms (3,674 cfu/L), *C. perfringens* (623 cfu/L), *A. hydrophila* (6,590 cfu/L), and *P. aeruginosa* (41 cfu/L). Treated water (tap water) at the filtration plant was free of any detectable indicator bacteria and free of human enteric viruses and in compliance with regulations. However, bacterial growth was observed in the water filtration units as previously reported.[18]

Study Population Characteristics

Table 1 reports the age and sex distributions of the two study groups at the study outset. By and large, the randomization produced very similar groups, although there were slightly more very young and fewer very old in the filtered water group than in the tap water group.

Over 95 percent of eligible families maintained their participation for Period 1 (Table 2). For the entire two periods, the participation rates were 86.0 percent in the tap water group and 92.6 percent in the filtered water group.

Water Consumption

Table 3 shows that at home the two groups consumed about the same amount of water, the tap water group consuming only tap water and the filtered water group consuming almost entirely filtered water. Away from home, both groups consumed mainly tap water and in both groups there was about five times as much water consumed at home than outside the home.

Disease Outcome

During the study period there were no outbreaks of gastrointestinal diseases officially reported to the local health authorities. The number of visits to physicians for gastrointestinal symptoms and of hospitalizations was similar in both groups.

Altogether 65 percent of families experienced at least one episode of HCGI symptoms during the entire period of observation and about 23 percent of individuals had at least one episode. The overall incidence of HCGI was 0.66 episodes per person per year and the mean duration of an episode was 1.9 days. The yearly incidence of HCGI symptoms was higher in Period 1 (Table 4A) than in Period 2 (Table 4B). Age was a strong determinant of incidence of HCGI symptoms with the highest rates in the under 5 age group.

Table 5 reports the incidence rates in the two groups adjusted to the covariate distribution of the tap water group. Three units of observation were analyzed: the family as a whole, the informant, and the youngest child within the family. Again, the pattern of difference between tap and filtered water groups was quite similar for the different units of observation and across the two periods. Using the entire family data, the preventable fraction was 35 percent in the Period 1 and 32.8 percent in the Period 2. Combining the two periods, the adjusted incidence rates were 0.76 in the tap water group and 0.50 in the filtered water group ($p < 0.01$).

The HCGI episode is a synthetic combination of a number of GI-related symptoms. Similar analyses were done for each of the symptoms included in the diary: the findings were very similar to what was observed for the synthetic HCGI variable.

Dose-Response Relationship

To determine whether increased tap water consumption in the home was associated with increased risk of morbidity, we restricted attention to the tap water group. Poisson regression analyses were carried out with the number of episodes in the entire second period as the dependent variable and the estimated number of glasses of water consumed per week as an independent variable. Table 6 shows that for both informants and youngest chil-

TABLE 2—Family Participation Patterns in the Two Study Groups across the Two Study Periods

Outcome	Filtered Water Number of Families	Tap Water Number of Families
Period 1—March to June 1988		
Total number enrolled	299	308
Quit—problems with filter	6	—
Quit—changed to filtered or bottled water	—	9
Quit—moved out of study area	5	3
Quit—unwilling to continue	3	1
Completed Period 1	285	295
Summer break		
Changed to ineligible[a]	2	9
Period 2—Sept 1988 to June 1989		
Total number enrolled	283	286
Quit—problems with filter	2	—
Quit—changed to filtered or bottled water	—	10
Quit—moved out of study area	3	5
Quit—unwilling to continue	1	6
Completed Period 2	277	265

[a] Most of these were due to a youngest child leaving home or reaching 18 years of age.

TABLE 3—Mean Number of Glasses of Water Consumed Weekly by Type of Water and by Group[a]

Type of Water Consumed	Group: Filtered Water	Group: Tap Water
At home		
Filtered water	37.4	0.0
Tap water	1.9	37.8
Away from home		
Filtered water	0.3	0.0
Tap water	7.7	8.7

[a] Based on a separate survey of the study population. Responses were obtained from 957 subjects in the filtered water group and 891 in the tap water group.

dren, there was an increasing risk with increasing consumption. Among the tap water group there was no relationship between the quantity of water consumed outside the home and morbidity. Among the filtered water group there was no relationship between the quantity of water consumed inside or outside the home and morbidity (results not shown).

Nature of the Excess

Analyses were also carried out to determine whether the higher incidence in the tap water group was due to more people experiencing symptoms or whether it was due to the same core of people experiencing more episodes. These analyses were restricted to those families for whom complete data were available for the entire study. As shown in Table 7, there was a slight difference between the two groups in the percentage of subjects with episodes, but the number of episodes per person affected was considerably higher in the tap water group than in the filtered water group. This implies that the main effect of tap water consumption was to increase the number of episodes among susceptibles.

Discussion

This randomized trial was carried out to determine whether drinking water conforming to currently accepted microbiological standards was the source of any excess of GI disease when compared with a group drinking the same water after a molecular filtration to remove contaminants. The study was carried out in a relatively homogeneous middle class suburban neighborhood, and the overall observed incidence of gastrointestinal symptoms was similar to incidence rates reported in other North American populations.[19–21] The difference between the periods is within range for this type of symptoms. There was, however, a clear and significant excess of HCGI symptoms among the tap water drinkers as compared with filtered water drinkers. This finding was bolstered by the relative consistency of the effect across different age, sex, subregion, and informant/proxy categories, and the observation that among tap water drinkers there appears to be an increasing risk with increasing consumption.

Before proceeding to speculate as to the reasons and impact of these findings, it is important to consider possible sources of bias, the main one being reporting bias. Although the intervention was randomized, study subjects were not blinded. The possibility of having a sham filter installed in homes of tap water subjects was considered, but it was not technically and financially feasible. Subjects with a sham filter would have perceived the inefficacy of their filter and would have recognized the characteristic chlorine odor of tap water.

It is conceivable that subjects participating in the tap water group but aware of the study objectives exaggerated their morbidity relative to the filtered water group. Similarly, subjects in the filtered water group, if they had faith in the quality of their water may have underestimated their morbidity. However, we doubt whether such biases had any significant effect. Symptoms such as vomiting and liquid diarrhea are not subjective and open to interpretation. If such biases were operating, it is likely that they would be most pronounced at the outset and diminish with time as the novelty of the health diary wore off. However, over the 15-month study period, the relative excess of GI disease was fairly constant, arguing against any such bias. Furthermore, the observation of a dose-response relationship is less plausibly attributable to such biases. As a general check on the credibility of the quality of reporting, it is of interest that the estimated incidence rate is similar to reports from other North American centers and the relative consistency of the effect across the different categories.[19–21]

Outside the home, study subjects from both groups consumed water from all kinds of sources. Thus there was some misclassification in labeling one group as tap water consumers and the other as filtered water consumers. Moreover, this would tend to attenuate the effect of any true difference due to domestic water source and thus reinforces the importance of the difference observed. Similarly, the finding of an apparent dose-response relationship in the tap water group is particularly impressive given the crudeness of the measure of dose which was based on a one-point-in-time survey. The treated water leaving the filtration point was free of bacterial pathogens and indicator bacteria as well as of cultivable human enteric viruses.

TABLE 4A—Incidence of HCGI Episodes for Period 1 for the Two Study Groups[a]

Subgroups	Filtered Water N	Filtered Water Rate[b]	Filtered Water S.E.[c]	Tap Water N	Tap Water Rate	Tap Water S.E.
Age group						
0–5	116	1.66	0.28	74	2.48	0.44
6–20	454	0.77	0.10	475	0.95	0.11
21–49	558	0.54	0.09	524	0.97	0.13
50+	85	0.31	0.25	135	0.46	0.24
Sex						
Male	613	0.75	0.09	609	0.88	0.10
Female	600	0.68	0.09	599	1.11	0.12
Informant/proxy						
Informant	298	0.65	0.13	304	1.00	0.16
Proxy	915	0.74	0.09	904	0.99	0.10
All subjects	1213	0.72	0.08	1206	0.99	0.10

[a] HCGI: highly credible gastrointestinal symptoms. See Methods Section for operational definition of HCGI and of an episode.
[b] Annual incidence per person.
[c] Standard error corrected for cluster nature of sampling.

TABLE 4B—Incidence of HCGI Episodes for Period 2 by Selected Population Subgroups[a]

Subgroups	Filtered Water N	Filtered Water Rate[b]	Filtered Water S.E.[c]	Tap Water N	Tap Water Rate	Tap Water S.E.
Age group						
0–5	115	1.13	0.18	72	1.59	0.27
6–20	428	0.46	0.07	427	0.66	0.08
21–49	512	0.39	0.06	458	0.56	0.08
50+	78	0.23	0.12	126	0.33	0.11
Sex						
Male	572	0.44	0.06	545	0.62	0.07
Female	561	0.52	0.07	538	0.66	0.08
Informant/proxy						
Informant	281	0.45	0.08	275	0.62	0.10
Proxy	852	0.49	0.06	808	0.65	0.07
All subjects	1133	0.48	0.06	1083	0.65	0.07

[a] HCGI: highly credible gastrointestinal symptoms. See Methods Section for operational definition of HCGI and of an episode.
[b] Annual incidence per person.
[c] Standard error corrected for cluster nature of sampling.

TABLE 5—Incidence of HCGI Episodes Derived from Poisson Regression Analysis, by Study Group for Each of Three Units of Observation[a]

Unit of Observation	Period 1 Filtered (n = 298)	Period 1 Tap (n = 304)	Period 1 Preventable Fraction (%)	Period 2 Filtered (n = 282)	Period 2 Tap (n = 276)	Period 2 Preventable Fraction (%)
Family	0.65	1.00**	35.0	0.43	0.64**	32.8
Informant	0.64	1.00*	36.0	0.44	0.63*	30.2
Youngest child	0.86	1.24*	30.7	0.56	0.80*	30.0

[a] Annual incidence per unit of observation, derived by Poisson regression with age, sex, and subregion in the models.
*Difference between the incidence rates is significant at $p < 0.05$
**Difference between the incidence rates is significant at $p < 0.01$

Public Health Impact

In the past decade, there has been growing concern about the safety of public water supplies. Much of this concern has focused on presumed noninfectious outcomes such as cancer and birth defects and, in this context, there is some fear that chlorination byproducts may be harmful.[10,22] Heretofore, it was assumed that current water quality standards were sufficient to protect the public against the risk of GI disease; the apparent lack of outbreaks seemed to support this view. Our results suggest that there is a non-trivial endemic level of unreported GI diseases due to the consumption of tap water; 35 percent of gastrointestinal illness was estimated to be attributable to the consumption of drinking water meeting current water quality standards.[5]

The main impact of tap water consumption seemed to be in increasing the numbers of episodes of susceptibles rather than in increasing the numbers of people with episodes. The severity of these illnesses was low and resulted in few demands on the health care system. The community studied is typical of many North American communities in terms of its water source being contaminated by sewage discharges, its modern water treatment facilities, its treatment procedures which are widely used, and its sociodemographic characteristics. Our results raise questions about the adequacy of current standards of drinking water quality, including source water quality, treatment procedures, and bacteriological quality of the water in the distribution system as well as in-house plumbing. ☐

Acknowledgments

The quality of the data gathered during this study was made possible only through the commitment of Denise Bourbonnais to whom the authors express their sincere appreciation. The authors are grateful to Jean Pellerin who set up the data management system and to Marie Désy who helped carry out the statistical analysis. The authors also acknowledge the skillful technical work of Nicole Filion, Louise Courtemanche, and Chantal Germain who were responsible for the virological and microbiological analyses. The authors are indebted to Dr. Al Dufour (USEPA, Cincinnati) and Dr. Richard Tobin (Environmental Health, Health and Welfare Canada, Ottawa) for their continuous support in the preparation and follow-up of this study. The help of the Ministry of the Environment of Quebec, through the use of laboratory facilities and expertise in the Montreal Laboratory, was also extremely important in the success of this study.

TABLE 6—Incidence of HCGI Episodes in Period 2 by Amount of Tap Water Consumed among Tap Water Subjects[a]

Unit of Observation/ Consumption	Number		Incidence[b]
Informant			
1–25 glasses/week	24		.42
26–45 glasses/week	92		.70
46+ glasses/week	114		.66
Trend test		p = .10	
Youngest child			
1–25 glasses/week	74		.55
26–45 glasses/week	92		.89
46+ glasses/week	64		1.10
Trend test		p = .10	

[a] Annual incidence per unit of observation during Period 2.
[b] Poisson regression model including age, sex, sub-region.

TABLE 7—Percent of Study Subjects Reporting HCGI Symptoms and Mean Number of Episodes per Unit of Observation in Both Periods Combined

	Group			
	Filtered Water (n = 272)		Tap Water (n = 262)	
Unit of Observation	% with Episodes[a]	Mean Number of Episodes[b]	% with Episodes	Mean Number of Episodes
Family	62.0	3.82	67.7	4.81
Informant	20.0	1.70	23.1	2.10
Youngest child	42.3	1.83	46.3	2.37

[a] Derived by logistic regression with covariables age, sex, geographic sub-region. Only subjects who had complete data sets in both periods are included in this table.
[b] Mean number of episodes among those subjects who reported at least one episode.

This study was funded by a grant from the National Health Research and Development Program (NHRDP) of the Ministry of Health and Welfare Canada.

References

1. World Health Organization: Guidelines for Drinking-Water Quality. Volume 1. Recommendations. 1984. Geneva: World Health Organization.
2. Payment P, Armon R: Virus removal by drinking water treatment processes. CRC Critical Reviews in Environmental Control 1989;19:15–31.
3. Smith HV, Rose JB: Waterborne cryptosporidiosis. Parasitology Today 1990;6:8–12.
4. Hayes EB, Matte TD, O'Brien TR, *et al.*: Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. New Engl J Med 1989;320:1372–1376.
5. Environmental Protection Agency: National interim primary drinking water regulations. 1976. EPA-570/9-76/003. Washington, DC: Office of Drinking Water Supply, U.S. Environmental Protection Agency.
6. Craun GF: Waterborne Diseases in the United States. Boca Raton, FL: CRC Press, 1985.
7. Isaacson M, Sayed AR: Health aspects of the use of recycled water in Windhoek, SWA/Namibia, 1974–1983. Diarrhoeal diseases and the consumption of reclaimed water. South Afric Med J 1988;73:596–599.
8. Fattal B, Guttman-Bass N, Agursky T, Shuval HI: Evaluation of health risk associated with drinking water quality in agricultural communities. Wat Sci Tech 1988;20:409–415.
9. Zmirou D, Ferley JP, Collin JF, Charrel M, Berlin J: A follow-up study of gastrointestinal diseases related to bacteriologically substandard drinking water. Amer J Public Health 1987;77:582–584.
10. Chang S-L: The safety of water disinfection. Ann Rev Public Health 1982;3:393–418.
11. Frome EL: The analysis of rates using Poisson regression models. Biometrics 1983;39:665–674.
12. McCullagh P, Nelder JA: Generalized Linear Models. London: Chapman & Hall, 1983;327–328.
13. Cox DR: The Analysis of Binary Data. London: Methuen, 1970.
14. Cox DR: Regression models and life tables (with discussion). J R Stat Soc B 1972;34:187–220.
15. Payment P, Gamache F, Paquette G: Microbiological and virological analysis of water from two water filtration plants and their distribution systems. Can J Microbiol 1988;34:1304–1309.
16. American Public Health Association, et al: Standard methods for the examination of water and wastewater, 16th ed. Washington, DC: APHA, AWWA, WPCF, 1985.
17. Ministère de l'environnement du Québec: Guide d'évaluation de la qualité bactériologique de l'eau potable. Québec: Editeur Officiel du Québec, 1981.
18. Payment P: Bacterial colonization of domestic reverse-osmosis water filtration units. Can J Microbiol 1989;35:1065–1067.
19. Hodges RG, McCorkle LP, Badger GF, Curtiss C, Dingle JH, Jordan WS Jr: A study of illness in a group of Cleveland families. XI. The occurrence of gastrointestinal symptoms. Amer J Hyg 1956;64:349–356.
20. Monto AS, Koopman JS, Longini IM, Isaacson RE: The Tecumseh Study: XII. Enteric agents in the community, 1976–1981. J Infect Dis 1983;148:284–291.
21. Monto AS, Koopman JS: The Tecumseh Study: XI. Occurrence of acute enteric illness in the community. Amer J Epidemiol 1980;112:323–333.
22. Crump KS, Guess HA: Drinking water and cancer: review of recent epidemiological findings and assessment of risks. Ann Rev Public Health 1982;3:339–357.

Risk Assessment and Control of Waterborne Giardiasis

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ABSTRACT

Background: Waterborne giardiasis has been increasing in the United States with 95 outbreaks reported over the last 25 years. The Safe Drinking Water Act has mandated control of this pathogen.

Methods: A risk assessment model was developed to estimate risk of infection after exposure to treated waters containing varying levels of *Giardia* cysts. The model was defined by a dose-response curve developed from human feeding studies for *Giardia* and assumed 2L of water consumption per day. Data on concentrations and distribution of the organism in source waters were used to assess exposure after varying reductions achieved through treatment.

Results: In surveys reporting prevalence and levels of *Giardia* cyst contamination, average levels of cysts in surface waters ranged from 0.33 to 104/100L; from pristine watersheds (protected from all human activity) 0.6 to 5/100L. Yearly risks were 4.8×10^{-3} for systems using polluted waters and 1.3×10^{-4} for pristine waters with a 10^{-3} treatment reduction.

Conclusion: Public Health officials will need to work with the water industry to ensure a risk of less than 1/10,000 for source waters with 0.7 to 70 cysts per 100 liters through treatment achieving reduction of 10^{-3} to 10^{-5}, respectively, of *Giardia* cysts. (*Am J Public Health.* 1991;81:709–713)

Introduction

Giardia is the most frequently isolated enteric protozoan from populations worldwide and the most common pathogenic parasite in the United States.[1] Waterborne giardiasis has been increasing in the United States with 95 outbreaks reported over the last 25 years,[2] and *Giardia* is the most common identifiable etiological agent of all waterborne outbreaks. Bennet *et al.*[3] have also estimated that 60 percent of all *Giardia* infections are acquired through contaminated water.

Giardia cysts may be found in water as a result of the deposition of fecal material from both man and animals. Surveys of *Giardia* cyst levels in various waters indicate that 26–43 percent of the surface waters were contaminated with *Giardia* cysts ranging in concentrations from 0.3 to 100 cysts per 100L.[5–10] Sykora[9] has reported an average of 10^4 cysts/100L in raw sewage with an approximate reduction of 10^{-2} after treatment (152 cysts/100 L).

The Surface Water Treatment Rule has been promulgated to address the amendments to the Safe Drinking Water Act for controlling *Giardia* in treated drinking water.[4] This rule mandates that all surface waters be treated to achieve at least a reduction of 10^{-3} (99.9 percent removal) of *Giardia* cysts. Disinfection is required for all systems and filtration is required unless the system meets site specific criteria and has a protected watershed. The US Environmental Protection Agency (EPA) has also recommended that a treatment be provided to ensure that populations are not subject to risk of infection of greater than 1:10,000 (10^{-4}) for a yearly exposure, and that this is an acceptable level of safety for potable waters.

As the new regulations and rules come into effect, health departments will be called upon to ensure that the public is adequately protected against waterborne disease. Risk assessment is a tool by which health officials can communicate with the water industry by interpreting water quality surveys and assisting in defining the adequacy of treatment adhering to EPA's recommendations of potable water quality and acceptable public health risks. This will become particularly important as states implement the Surface Water Treatment Rule, evaluate new technologies, and determine what water management practices will impact public health.

This paper presents a risk assessment model that was used to estimate risk of infection due to waterborne exposure to *Giardia*. Dose-response curves were developed based on human infectivity studies, and data on the occurrence of *Giardia* cyst contamination in waters throughout the US were used to define the water treatment needed to reduce the risk of waterborne giardiasis.

Methods

To predict a potential public impact when the risk is small, models are used to estimate the risk after exposure. Haas reviewed three probability models for their ability to describe experimental dose-response data for humans after exposure to

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various enteric microorganisms.[11] Using the same approach, the simple exponential and beta distributed effectiveness models were evaluated for prediction of *Giardia* infectivity using experimental data developed by Rendtorff[12,13] in human feeding studies.

In Rendtorff's experiments, *Giardia* cyst doses ranging from 1 to 10^6 were fed to volunteers, and a positive response was measured by cyst excretion in the feces (Appendix). Laboratory dose-response studies generally appear to be conducted under conditions where the distribution of microorganisms in the administered dose may be regarded as Poisson. Under these conditions, if one microorganism is sufficient to cause an infection, and if host-microorganism interactions are constant, then the probability of an infection (P_i) resulting from ingestion of a single volume of liquid containing an average number of organisms (N) may be defined by a simple exponential equation.

$$P_i = 1 - \exp(-rN)$$

In this equation, r is the fraction of microorganisms that are ingested which survive to initiate infections ("host-microorganism interaction probability") (Appendix). In this particular case, the exponential model was statistically consistent with the Rendtorff data and r was calculated to equal −0.01982.

The 95 percent confidence limits to the parameter r in the exponential model were computed using a likelihood ratio technique. The resulting interval estimate for r is 0.009798–0.03582. This range was used in the preparation of Figure 1.

Exposure Estimates

Surface waters were classified into two categories: polluted waters contaminated by sewage and agricultural discharges; and pristine waters originating from protected watersheds without point source pollution or input from human activities. *Giardia* cyst levels were examined for the peak level of contamination from a single sample, average concentrations for each site, and average cyst levels for each water classification. Geometric means were calculated from the average concentrations from each site.[5–10] *Giardia* cyst levels were calculated as cysts/100 L as large volumes of water are routinely sampled for determining levels of contamination.

Using the exponential model, the potential risk of infection was determined with varying levels of *Giardia* cysts in drinking water. The model assumed the consumption of two liters of water per day, and exposure N in the formula was defined by numbers of cysts per liter times two liters. Levels of *Giardia* cysts found in polluted and pristine source waters and assumed levels of 10^{-3}, 10^{-4}, and 10^{-5} removal by treatment were used to estimate exposure in the model. Maximum daily risk was estimated using the peak level of contamination and yearly risk was determined using the geometric mean concentration of *Giardia* cysts for 365 days of exposure. Probability of infection was determined assuming a Poisson distribution of microorganisms in the drinking water,[11] i.e.,

> risk of contracting one or more infections = $1-(1-\Pr(N))^x$ where x = the number of days exposed and $\Pr(N)$ = the daily risk using the geometric average for N.

Data from five waterborne outbreaks of giardiasis were also evaluated for the *Giardia* cyst level of contamination detected in the drinking water and the attack rates in the exposed population.[14] This information was compared to the estimated infection rates developed by the exponential model after varying days of exposure.

Results

To ensure less than a daily risk of 10^{-4}, systems using source waters containing 250, 2,500 and 25,000 cysts per 100L would need to reduce the level of *Giardia* cysts by 10^{-3}, 10^{-4} and 10^{-5}, respectively, through drinking water treatment (Table 1). Examples of yearly risks for exposure to varying levels of *Giardia* cysts in drinking water are summarized in Table 2. To ensure less than a yearly risk of 10^{-4}, systems using source waters containing a geometric concentration of 0.7, 7.0 and 70 cysts per 100L would require a reduction of 10^{-3}, 10^{-4} and 10^{-5}, respectively, by treatment.

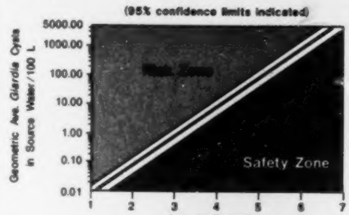

FIGURE 1—Log 10 Treatment Reductions for *Giardia* Cysts in Source Waters Yielding 10^{-4} Annual Risk (95% confidence limits indicated)

TABLE 1—Estimated Daily Risk of *Giardia* Infections from Various Levels of Cyst Contamination in Drinking Water Using an Exponential Risk Assessment Model

Worst Daily Risk Pr(N)	Cyst Exposure	Maximum Cyst Concentration in Finished Water per 100L	Maximum Cyst Concentration in Source Water Per Level of Treatment Reductions, Cysts/100L: 10^{-3}	10^{-4}	10^{-5}
$10^{-3.5}$	0.015	0.75[a]	7.5×10^2	7.5×10^3	7.5×10^4
10^{-4}	0.005	0.25	2.5×10^2	2.5×10^3	2.5×10^4
$10^{-4.5}$	0.0015	0.075	75	7.5×10^2	7.5×10^3
10^{-5}	0.0005	0.025	25	2.5×10^2	2.5×10^3

[a] Levels of cysts detected during waterborne outbreaks of giardiasis.

In surveys reporting prevalence and levels of *Giardia* cyst contamination in surface waters, average levels of cysts ranged from 0.33 to 104/100L. Eight of the areas where samples were collected from received treated sewage or agricultural discharges, however, the level of pollution was not documented.[5–10] In water samples originating from pristine watersheds (protected from all human activity) *Giardia* cyst levels averaged 0.6 to 5/100L. The percentage of positive samples did not vary dramatically from polluted to pristine waters (43 percent and 35 percent, respectively). Peak contamination in a single sample was 5.5 times higher from polluted waters than from pristine waters. However, geometric averages of cysts were 50 times higher for samples collected from polluted waters versus those collected from pristine waters.

The majority of data for the pristine waters was developed from a study by

TABLE 2—Estimated Yearly Risk of *Giardia* Infections from Various Levels of Cyst Contamination in Drinking Water Using an Exponential Risk Assessment Model

Yearly Risk $1-(1-Pr^a(N))^{365}$	Average Daily Cyst Exposure for a Year	Geometric Mean Cyst Concentration in Finished Water per 100L	Geometric Mean Cyst Concentration in Source Water Per Level of Treatment Reductions 10^{-3}	10^{-4}	10^{-5}
			Cysts/100 L		
$10^{-3.5}$	4×10^{-5}	2×10^{-3}	2.0	20	200
$10^{-4.0}$	1.4×10^{-5}	7×10^{-4}	0.7	7.0	70
$10^{-4.5}$	4×10^{-6}	2×10^{-4}	0.2	2.0	20
10^{-5}	1.4×10^{-6}	7×10^{-5}	0.07	0.7	7.0

^{a}Pr = Daily Probability of Risk

TABLE 3—Potential Levels of Cysts in Treated Drinking Water and Probability of Infection

Source Water	Reduction by Treatment	Geometric Cyst Levels per 100L in Drinking Water	Yearly Risk^b	With Peak Contamination Cysts/100L in Drinking Water	Daily Risk
Polluted 33 cysts^a/100L	10^{-3}	0.033	4.8×10^{-3}	0.625	2.5×10^{-4}
	10^{-4}	0.0033	4.8×10^{-4}	0.062	2.5×10^{-5}
	10^{-5}	0.00033	4.8×10^{-5}	0.006	2.5×10^{-6}
Pristine 0.9 cysts^a/100L	10^{-3}	0.0009	1.3×10^{-4}	0.114	4.6×10^{-5}
	10^{-4}	0.00009	1.3×10^{-5}	0.0114	4.6×10^{-6}
	10^{-5}	0.000009	1.3×10^{-6}	0.0011	4.6×10^{-7}

^{a}geometric average
^{b}Risk = $1-(1-Pr(N))^{365}$

Ongerth, *et al.*[6] The Tolt, Green and Cedar watersheds in the State of Washington were extensively sampled with 222 samples collected. The averages reported in the publication included a 22 percent recovery adjustment for method efficiencies. The data from the Ongerth study[6] used in this paper did not include such adjustments since the data used from other studies[5,7–10] did not take into account adjustments for recovery efficiencies.

Daily and yearly risks were developed for the peak cyst level and the geometric average, respectively, for the two water categories (Polluted and Pristine). The data are shown in Table 3. Yearly risks ranged from 4.8×10^{-3} for systems using polluted waters with a 10^{-3} treatment reduction to 1.3×10^{-6} for pristine waters with a 10^{-5} treatment reduction. Between 10^{-4} and 10^{-5} reduction with treatment would be required for polluted waters to achieve a similar risk as pristine waters treated for a 10^{-3} removal of *Giardia* cysts. For daily risks for waters containing peak levels of *Giardia* cysts, the differences were not as dramatic between systems using polluted versus pristine source waters. The peak daily risk was approximately five times greater in systems using polluted versus pristine waters receiving similar treatment for removal of cysts.

Water samples were collected and *Giardia* cyst levels were determined during the investigation of five waterborne outbreaks of giardiasis. Attack rates varied from a low of 0.5 percent in the Houtzdale and Pittston outbreaks to a high of 16 percent in the Ft. Plain outbreak. The level of cyst contamination ranged from 0.6 to 21 cysts/100L. Generally the lower levels of contamination were associated with lower levels of infection in the population. The data are shown in Table 4.

All five outbreaks were associated with unfiltered chlorinated surface waters. Three factors primarily influenced the attack rate for infection: first, the level of contamination; second, the level of cyst viability and inactivation through chlorination; and third, the length of exposure to the population whose prior exposure to *Giardia* (and potential immunity) was unknown. Much of this detail is not known. The exponential risk model was applied to the known cyst levels from the outbreak and infection rates were estimated for varying days of exposure. It is unlikely that there was a single day of exposure or that the exposure was equal to the duration of the outbreaks. If one uses intermediate times of exposure, five to 10 days, the infection rates developed by the model ranged from 0.1 to 7.7 percent.

Discussion

Many regulatory and public health agencies have accepted risk assessment models to evaluate the importance of chemical pollutants in water. How well these models reflect reality depends on the accuracy of the model for characterizing the independent variables, assumptions, and the data used to develop the dose-response curves and exposure.

The most essential component to the risk model described in this paper is the dose-response curve. *Giardia* species/strains are known to have a low infectious dose. Rendtorff and Holt demonstrated in 1954 that ingestion of as few as 10 cysts was capable of initiating infection in two volunteers. It must be kept in mind that infection was measured by cyst excretion and illness was not determined. Asymptomatic *Giardia* infections may range between 39 percent and 76 percent for children less than five years of age and adults, respectively.[15,16] Symptomatic infections, however, have been reported at a rate of 50 percent to 67 percent and as high as 91 percent.[17] Chronic giardiasis may also develop in as many as 58 percent of the population infected.[18] Thus the illness to infection ratio is highly variable. We addressed only infection in this analysis of risk.

Another important issue regarding the dose response curve based on the Rendtorff data is uncertainty about infectivity due to strain variation and the immune response to infection by different populations. The Rendtorff data are derived from one *Giardia lamblia* strain and one relatively small sample population. The confidence interval around the probability of infection does not take these uncertainties into account when using the model as a predictive tool.

Assuming the dose response relationship derived from the Rendtorff data is representative, we may be overestimating

TABLE 4—*Giardia* Cyst Contamination of Drinking Waters during Waterborne Outbreaks of Giardiasis and the Relationship to an Exponential Risk Model

		Outbreak Data		Estimated Infection Rates[b] after Exposure for (Days)				
Outbreak	Duration (days)	*Giardia*[a] Cysts/100L	Attack Rates %	1	5	10	30	60
Pittsfield	30	21	3	0.84	3.9	7.7	21	38
Ft. Plain	60	19.5	16	0.78	3.8	7.5	21	37
Houtzdale	?	2.4	0.5	0.09	0.4	0.9	2.7	5.2
Pittston	?	0.87	0.5	0.03	0.1	0.3	0.9	1.8
Wilkes-Barre	?	11	9	0.4	2.0	3.9	11.3	21
		0.6	1	0.02	0.1	0.2	0.6	1.2

[a]Data received from Dr. Judith Sauch, Environmental Protection Agency, Cincinnati, OH.[14,15]
[b]Estimated from the exponential risk model.

risks based on the assumption that all cysts are viable and all cysts in water are species which infect humans. In addition, the 2L of tap water consumption may represent overestimation of exposure, depending on individuals consumption of other beverages. However, the underestimation of risk may be of greater concern due to underestimation of exposure by the inefficiencies of the methods for detection of *Giardia* cysts in water.

In spite of its limitations, the model can be used to examine waterborne outbreak data and disease surveillance data associated with various exposure routes. Epidemiological data, may be even more insensitive and inaccurate. Attack rates for waterborne outbreaks, defined by the number of illnesses in the exposed population, are developed using a variety of methods; they do not take into account unreported cases or asymptomatic infections, and may include individuals who were not exposed. Estimation of infection based on attack rates is probably understated.

Levels of cysts ranging from 0.6 to 21/100L detected in waters were associated with outbreaks of disease. *Giardia* cyst levels found in drinking waters averaged 0.19/100L during non-outbreak conditions and were rarely above 1/100L.[19] Based on the data in Table 4, it appears the model may be useful in estimating probability of infection. The greatest limitation of the model may be the underestimation of infection and disease through the use of cyst concentrations in water without taking into account method efficiencies, peak contamination levels, and duration of exposure.

By applying information on *Giardia* cyst levels to Figure 1, public health agencies, regulatory agencies, and water utilities can evaluate the adequacy of current treatment of a drinking water supply, the need for more effective treatment, or the type of treatment needed in developing new water supplies.

Public health workers must not only have an understanding of waterborne disease. The implications of new regulations, watershed protection, and water treatment on indigenous potential waterborne infections in the population must also be understood. Standard methods are available to assess *Giardia* cyst contamination in water. Health agencies can utilize such data in a risk assessment, cost-benefit approach to establish rational policies for public health protection. ☐

Acknowledgments

This work was initiated during the authors summer term as an Environmental Science and Engineering Fellow sponsored by the American Association for the Advancement of Science at the Office of Drinking Water, US Environmental Protection Agency, Washington, DC, 1988.

References

1. Feachem RG, Bradley DH, Garelick H, Mara DD: Sanitation and Disease Health Aspects of Excreta and Wastewater Management. New York: John Wiley and Sons, 1983.
2. Craun GF: Surface water supplies and health. J Am Water Works Assoc 1988; 80:40–52.
3. Bennett JV, Homberg SD, Rogers MF, Solomon SL: Infectious and parasitic diseases. Am J Prev Med 1987; 3:102–114.
4. National Primary Drinking Water Regulations: Filtration Disinfection; Turbidity, *Giardia lamblia*, Viruses, Legionella, and Heterotrophic Bacteria. Final rule, 40 CFR parts 141 and 142. Federal Register June 29, 1989; 54:27486–27541. Washington, DC: Govt Printing Office, 1989.
5. Hibler CP: Analysis of municipal water samples for cysts of *Giardia*. *In:* Wallis P, Hammon B (eds): Advances in *Giardia* Research: University of Calgary Press, 1988; 237–245.
6. Ongerth JE: *Giardia* cyst concentrations in river water. J Am Water Works Assoc 1989; 81:81–86.
7. Rose JB, Kayed D, Madore MS, Gerba CP, Arrowood MJ, Sterling CR: Methods for the recovery of *Giardia* and *Cryptosporidium* from environmental waters and their comparative occurrence. *In:* Wallis P, Hammond B (eds): Advances in *Giardia* Research: University of Calgary Press, 1988; 205–209.
8. Rose JB, Darbin H, Gerba CP: Correlations of the Protozoa, *Cryptosporidium* and *Giardia* with water quality variables in a watershed. Water Sci Tech 1988; 20:271–276.
9. Sykora JL, States SJ, Bancroft WD, Boutrous SN, Shapino MA, Conley LF: Monitoring of water and wastewater for *Giardia*. *In:* Advances in Water Analysis and Treatment Water Quality Technology Conference, Portland, OR, 1986; 1043–1054. Available from American Water Works Assoc, Denver, CO.
10. Willey BR, Harmon DJ, Benjes HH Jr: Survey and evaluations of 80 public water systems in Wyoming. Culp/Wesner/Culp Consulting Eng, Denver, CO. Prepared for US Environmental Protection Agency, Region VIII National Environmental Health Association: January 1986.
11. Haas CN: Estimation of risk due to low doses of microorganisms: A comparison of alternative methodologies. Am J Epidemiol 1983; 118:573–582.
12. Rendtorff RC: The experimental transmission of human intestinal protozoan parasites. II. *Giardia lamblia* cysts given in capsules. Am J Hyg 1954; 59:209–220.
13. Rendtorff RC, Holt CJ: The experimental transmission of human intestinal protozoan parasites. IV. Attempts to transmit *Endamoeba coli* and *Giardia lamblia* by water. Am J Hyg 1954; 60:327–328.
14. Kent GP, Greenspan JR, Herndon JL, Mofenson LM, Harris JS, Eng TR, Waskin HA: Epidemic giardiasis caused by a contaminated public water supply. Am J Public Health 1988; 78:139–143.
15. Craft CJ: *Giardia* and giardiasis in childhood. Pediatr Infect Dis 1981; 1:196–211.
16. Wolf MS: Managing the patient with giardiasis: Clinical, diagnostic and therapeutic aspects. *In:* Jakubowski W, Hoff JC (eds): Waterborne Transmission of Giardiasis. Cincinnati, OH: EPA, 1979; 39–52.
17. Veazie L, Brownlee I, Sears HJ: An out-

break of gastroenteritis associated with *Giardia lamblia*. *In:* Jakubowski W, Hoff JC (eds): Waterborne Transmission of Giardiasis. Cincinnati, OH: EPA, 1979; 174–191.

18. Chester AC, MacMurray FG, Restifo MD, Mann O: Giardiasis as a chronic disease. Digest Dis Sci 1985; 30:215–218.
19. Rose JB: Environmental sampling for waterborne pathogens: Overview of methods, application, limitations and data interpretation. *In:* Craun G (ed): Investigation of Waterborne Disease Outbreaks. Arlington, MA: Eastern Research Group, Inc, 1990.

APPENDIX

For the purpose of modeling, it was assumed that the cysts have a random distribution in water and that a Poisson distribution would govern the probability of any given exposure.[11] The risk of infection was defined as a function of the exposure (N organisms) and the interaction of the host and pathogen. The parameters characterizing this interaction (r) were determined using the human infectivity data set. The data set for the experimental infection and development of the model areshown in Table A-1. The single-hit exponential equation for the probability of infections is shown in Table A-2. The constant characterizing the host-cyst interaction with the best fit values to the data set is also presented. An average r value (that fraction of microorganisms that are ingested which survive to initiate infection) for the exponential model was computed by determining the value of r at each dose. The chi square goodness of fit test was used to evaluate the appropriateness of the model for the data set.The single hit exponential model was used toevaluate the risk of waterborne giardiasis. Low level contamination of drinking water with *Giardia* (0.5 cysts/100L)would result in a potential infection rate of 0.02 percent in the exposed population using the exponential model while at high levels of contamination, (100 cysts/100L) a 3.9 percent infection rate was determined (Table A-3).

TABLE A-1—Data Set for Experimental Infection for *Giardia* Cyst Exposure

Cyst Dose	Volunteers' Response: Positive[a]	Volunteers' Response: Negative	Percent with Positive Response	Predicted Number of Positives[b]
1	0	5	0	0.098
10	2	0	100	0.359
25	6	14	30	7.809
10^2	2	0	100	1.724
10^4	3	0	100	3
10^5	3	0	100	3
3×10^5	3	0	100	3
10^6	2	0	100	2

[a] Positive response denoted by cyst excretion.
[b] Based on function p = 1−exp(−0.0196N)
Source: Rendtorff[12,13]

TABLE A-2—Best Fit Distribution Parameters for *Giardia* Infection Probability

Model	*Exponential Model*
Equation	p = 1 − exp(−rN)
Constant(s)	r = (−.01982)
Expected probability of infection from an average dose of 1 cyst	0.0198
Number of infections in a population of 100, each exposed to 1 cyst	2

[a] 95% confidence intervals for r = 0.009798–0.03582.
Source: Haas[11]

TABLE A-3—Potential Risk of Infection in *Giardia* Contaminated Drinking Water Using an Exponential Risk Assessment Model

Giardia concentration in water: Cysts/100L	*Giardia* concentration in water: Cysts/L[a]	Daily Risk	Percent Infection
0.5	0.005	2.0×10^{-4}	0.02
0.8	0.008	3.2×10^{-4}	0.032
1	0.01	4.0×10^{-4}	0.04
5	0.05	$2\ \times10^{-3}$	0.2
10	0.1	4.0×10^{-3}	0.4
15	0.15	6.0×10^{-3}	0.6
20	0.2	8.0×10^{-3}	0.8
50	0.5	2.0×10^{-2}	2.0
100	1	3.9×10^{-2}	3.9
200	2	7.7×10^{-2}	7.7

[a] Assume consumption of two liters of drinking water per day.

Lyme Disease: A Proposed Ecological Index to Assess Areas of Risk in the Northeastern United States

ABSTRACT

Background: **Recent public awareness has resulted in a demand for information about ways to reduce the risk of acquiring Lyme disease.**

Methods: **Twenty-two school properties and recreational areas within a Lyme disease endemic area of central Monmouth County, New Jersey were evaluated for risk of transmission using an ecological index on the suitability, amount, and access to *Ixodes dammini* habitat by target human populations and the abundance of infected adult ticks.**

Results: **The characterization of tick habitat accurately predicted the elimination of 11 sites from concern. Of the remaining 11 sites, six were classified high risk and five as moderate risk. On-site tick surveys identified infected *I. dammini* adults at only four sites (three risk; one moderate risk).**

Conclusions: **These results indicate that the use of selected ecological parameters provides a cost-effective method to rapidly identify areas at risk for Lyme disease transmission. (*Am J Public Health.* 1991;81:714–718)**

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Introduction

The extensive media attention recently afforded to Lyme disease has resulted in a dramatic increase in public awareness and demand for information regarding this tick-borne, spirochete-induced illness. The public is concerned about ways to reduce the risk of acquiring Lyme disease, including the relative safety of discrete geographical areas such as schools, recreational areas, and residential communities.

Early ecological studies in New Jersey identified areas of potential risk at the regional level through deer surveys[1] and serosurveys of canines.[2] As Lyme disease became more prevalent, areas at risk were determined by plotting case reports.[3] While local health officials could identify general areas at risk by randomly conducting tick surveys or retrospectively plotting case reports, a cost-effective method to prospectively identify specific geographical areas at risk for Lyme disease was unavailable. We propose here an index for use by public health and allied agencies which systematically assesses specific geographical areas for potential and actual risk of Lyme disease transmission through evaluation of key ecological parameters.

Methods

Study Sites

In response to increased public inquiries, the Freehold Township Health Department petitioned the New Jersey State Department of Health for assistance in evaluating the relative risk of Lyme disease transmission at all eight public school properties and 14 recreational areas within its jurisdiction. The public school properties ranged in size from 4.83 to 69.59 acres ($\bar{x}$ = 19.80 acres). The recreational areas ranged in size from 0.5 to 200 acres ($\bar{x}$ = 40.5 acres).

Identification of Tick Habitat

The northern deer tick, *Ixodes dammini* Spielman, Clifford, Piesman and Corwin is most prevalent in wooded areas, associated ecotones, and old fields in secondary woody succession.[4] Extensive surveys of potential tick habitats common to the Inner and Outer Coastal Plains of New Jersey where the majority of Lyme disease cases are reported[3] confirmed these associations (unpublished data). In upland areas of the coastal plain physiographic regions of New Jersey and other northeastern states, habitats generally yielding the highest populations of *I. dammini* are comprised of mixed hardwood, mixed hardwood/pine, and pine forests with a dense shrub layer dominated by highbush blueberry, *Vaccinium corymbosum* L.; lowbush blueberry, *Vaccinium angustifolium* Ait.; and huckleberries, *Gaylussacia* spp. with some mountain laurel, *Kalmia latifolia* L.; bayberry, *Myrica pensylvanica* Loisel; and catbrier or greenbrier, *Smilax* spp.[5]

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TABLE 1—Summary of *I. dammini* Abundance and Infection Rates from Recreational Areas Yielding Ticks in Freehold Township, New Jersey

Surveyed Area	Number of Adult Ticks (60-Minute Survey)	Rate of Infection by *B. burgdorferi* (%)
A	48	41.7
B	88	57.1
C	28	50.0
D	24	33.3
NWS Earle	74–180	41.9

Tick Surveys

Tick surveillance would normally be scheduled only for those sites identified as having high or moderate risk of potential transmission based on the evaluation of habitat. To test the efficacy of the proposed index, all 22 sites were surveyed for the presence of ticks. Tick populations were evaluated during the period of peak adult activity in the fall by walking throughout suitable tick habitat at each of the subject sites for a 60-minute period.[4,6] At smaller sites, the survey time was reduced and the number of ticks collected was extrapolated to the 60-minute survey period for ease in comparison. This recognized survey technique provides an accurate representation of the expected number of adult ticks to be encountered by humans.[7] When no suitable habitat was present, the site was then randomly walked. Ticks adhering to investigators' light-colored, cotton coveralls were removed and subsequently evaluated for the presence of spirochetes using dark-field microscopy.[8] Based on previous studies in Monmouth County, the spirochetes identified in this study were assumed to be *Borrelia burgdorferi*.[9]

Ecological Index

The proposed ecological index is designed to use information on the type of plant communities and abundance of infected adult *I. dammini* to rapidly predict the risk of Lyme disease transmission at discrete geographical sites. Although plant communities may be assessed throughout the year, the seasonality of adult tick activity would naturally restrict the use of the proposed index in New Jersey to early spring and fall.[10] To alleviate this problem, the index was divided into two phases. Phase I permits the characterization of a site with respect to potential risk of transmission in the absence of tick activity by evaluating the suitability, amount, and accessibility of *I. dammini* habitat. Point values ranging from a minimum of 1 to a maximum of 5 are assigned to each of the three ecological parameters and totaled. The resulting score is used to select the appropriate response action for each site and determine the need to proceed to Phase II of the proposed index.

Rather than sole reliance on habitat to predict the risk of transmission at a particular site, Phase II of the proposed index is designed to utilize, in conjunction with the information on habitat, data on the presence and size of the deer tick population, and rate of *B. burgdorferi* infection. Point values of 1 to 5 are assigned to each of the five ecological parameters and totaled to achieve a final score for each site. This scoring procedure is used to categorize actual risk and establish priorities for intervention strategies.

Detailed descriptions of the ecological parameters, scoring procedures, and response actions for both phases of the proposed ecological index along with the survey form are provided in the Appendix in an instructional format.

Survey Procedure

The assessment of potential risk of transmission for each of the subject sites was performed on June 22, 1989 and July 19, 1989. To determine actual risk of transmission, the subject sites were surveyed for *I. dammini* between October 27, 1989 and November 8, 1989. These dates were well within the period of peak activity for *I. dammini* adults in New Jersey.[10] Surveys were performed only on fair days between 1000–1400 hours when peak diurnal activity was expected. Ticks collected during walking surveys were retained for subsequent analysis. Data on tick abundance and infection rate were recorded on the original survey form for each site. The sites were then ranked with respect to risk of transmission and need for intervention. To document that temporal and meteorological conditions were conducive to tick questing, a site at nearby Naval Weapons Station Earle (NWS Earle), Colts Neck, New Jersey exhibiting similar habitat and known to have consistently high populations of infected *I. dammini*[9] was also surveyed for ticks on each of the study dates.

Results

Potential Risk of Transmission

Of the eight public school properties, only one demonstrated high risk of transmission. Moderate risk of transmission was assigned to one school, while the remaining six schools received a low risk rating.

Regarding the recreational areas, five of 14 were considered to have high risk of transmission ratings, while four sites were assigned moderate risk of transmission ratings. The remaining five recreational areas received low risk of transmission ratings.

Actual Risk of Transmission

None of the eight school properties surveyed yielded *I. dammini* adults. On the survey days, the site at NWS Earle produced 88 and 114 ticks per 60-minute survey, confirming tick activity on the survey dates. Of the eight schools, one was classified as limited risk, three as no present risk, and four as no risk likely.

Ticks were collected from only four recreational areas. In 60-minute surveys, the four sites yielded between 24 and 88 adult ticks. At the NWS Earle site, 74, 88, and 180 ticks were collected in equivalent 60-minute surveys, again confirming tick activity. Minimum field infection rates at the four recreational sites ranged between 33.3 percent and 57.1 percent. During the survey period, 41.9 percent of adult ticks collected from NWS Earle were infected with spirochetes. The tick collection and infection rate data for sites yielding ticks are summarized in Table. 1. As a result of tick surveys, two of the 14 recreational sites were classified as definite risk, three as potential risk, one as limited risk, three as no present risk, and the remaining five as no risk likely.

Discussion

The proposed ecological index was designed for use in upland areas of the coastal plain physiographic regions of the northeastern United States where the majority of Lyme disease cases occur.[11] In this investigation, the proposed index provided a rapid, systematic, and cost-effective method of eliminating from public health concern high use public property by assessing the ability of existing plant communities to serve as habitat for *I. dammini*. Once significant potential risk was determined by surveys of vegetation, the actual risk of transmission and appropriate response action were determined based on the abundance of infected adult deer ticks. Where the response action may include chemical control, the proposed index will assist the user in identifying areas requiring treatment, thereby reducing unnecessary acaricide applications.

Although tested on only 22 sites comprising 725 acres within a hyperendemic area of New Jersey, we believe that, with minor modifications, the proposed index can be used in other ecosystems and for other tick vectors. For example, the "suitability of habitat" parameter can be modified to reflect differences in vegetation utilization by deer ticks in the ecosystem under investigation. Although we found the collection of adult ticks to be the most cost-effective method in characterizing the deer tick population, other survey techniques may be substituted to evaluate subadult *I. dammini* or other tick vectors. In such cases, criteria of the "tick abundance" and "tick infection rate" parameters will require modification. The proposed ecological index is designed to be a flexible survey instrument that provides public health officials and others with a means to rapidly assess the potential and actual risk of Lyme disease transmission at various sites within their jurisdictions. □

References

1. Schulze TL, Lakat MF, Bowen GS, Parkin WE, Shisler JK: *Ixodes dammini* (Acari: Ixodidae) and associated ixodid ticks collected from white-tailed deer in New Jersey, USA: Geographical distribution and relation to selected environmental and physical parameters. J Med Entomol 1984; 21:741–749.
2. Schulze TL, Bosler EM, Shisler JK, Ware IC, Lakat MF, Parkin WE: Prevalence of canine Lyme disease from an endemic area as determined by serosurvey. Zbl Bakt Hyg 1986; A263:427–434.
3. Bowen GS, Schulze TL, Parkin WE: Lyme disease in New Jersey 1978–1982. Yale J Med Biol 1984; 57:661–668.
4. Carey AB, Krinsky WL, Main AJ: *Ixodes dammini* (Acari: Ixodidae) and associated ixodid ticks from south-central Connecticut, USA. J Med Entomol 1980; 17:89–99.
5. Robichaud B, Buell MF: *Vegetation of New Jersey*. New Brunswick, NJ: Rutgers University Press, 1973; 340 pp.
6. Schulze TL, Bowen GS, Lakat MF, Parkin WE, Shisler JK: The role of adult *Ixodes dammini* (Acari: Ixodidae) in the transmission of Lyme disease in New Jersey, USA. J Med Entomol 1985; 22:88–93.
7. Ginsberg HS, Ewing CP: Comparison of flagging, walking, trapping, and collecting ticks from hosts as sampling methods for northern deer ticks, *Ixodes dammini*, and lone-star ticks, *Amblyomma americanum* (Acari: Ixodidae). Exp Appl Acarol 1989; 7:313–322.
8. Bosler EM, Coleman JL, Benach JL, Massey DA, Hanrahan JP, Burgdorfer W, Barbour AG: Natural distribution of the *Ixodes dammini* spirochete. Science 1983; 220:321–322.
9. Schulze TL, Lakat MF, Parkin WE, Shisler JK, Charette DJ, Bosler EM: Comparison of rates of infection by the Lyme disease spirochete in selected populations of *Ixodes dammini* and *Amblyomma americanum* (Acari: Ixodidae). Zbl Bakt Hyg 1986; A263:72–78.
10. Schulze TL, Bowen GS, Lakat MF, Parkin WE, Shisler JK: Seasonal abundance and host utilization of *Ixodes dammini* (Acari: Ixodidae) and other ixodid ticks from an endemic Lyme disease focus in New Jersey, USA. J Med Entomol 1985; 23:105–109.
11. Centers for Disease Control: Lyme disease surveillance summary 1990. Atlanta, GA: CDC, 90-1(1).

APPENDIX
Lyme Disease Survey Form
Site ____________________
Investigator(s)
Potential Risk ____________________ **Date** ____________
Actual Risk ____________________ **Date** ____________

1. Suitability of Habitat: 1 2 3 4 5
Comments ____________________

2. Amount of Habitat: 1 2 3 4 5
Comments ____________________

3. Accessibility of Habitat: 1 2 3 4 5
Comments ____________________

4. Tick Abundance: 1 2 3 4 5
Comments ____________________

5. Tick Infection Rate: 1 2 3 4 5
Comments ____________________

Final Score:
Potential Risk of Transmission ____________
Response Action ____________________

Actual Risk of Transmission ________/________/________ = ________
Response Action ____________________

(continued)

APPENDIX continued
Instructions for Use of Lyme Disease Survey Form

Index Parameters

Phase I of the ecological index prospectively assesses discrete geographical sites for the potential risk of Lyme disease transmission by evaluating three ecological parameters. In the absence of tick data, point values are assigned to reflect the degree to which each parameter enhances the likelihood of transmission. In addition to these three parameters, Phase II employs tick abundance and tick infection rate to categorize these sites for the actual risk of Lyme disease transmission. Point values were similarly assigned to these parameters to complete the assessment.

Parameter Number	Parameter Description	Point Value	
		High	Low
1	Suitability of Habitat	5	1
2	Amount of Habitat	5	1
3	Accessibility of Habitat	5	1
4	Tick Abundance	5	1
5	Tick Infection Rate	5	1

Description of Parameter

1. Suitability of Habitat

This parameter addresses the suitability of the habitat (plant community) at the subject site for supporting populations of *Ixodes dammini* ticks.

Point Value	Habitat Description
5	= mixed hardwoods or mixed hardwoods/pine forest with substantial (>75% coverage) shrub layer dominated by highbush blueberries, lowbush blueberries, and huckleberries with some laurels, bayberries, catbrier, saplings/seedlings of the aforementioned tree species, and associated ecotones (Type 1 habitat).
4	= same as above except with a modest (25%–50% coverage) shrub layer (Type 2 habitat).
3	= same as above except with a sparse (<25% coverage) shrub layer or fields in secondary woody succession (Type 3 habitat).
2	= meadows, overgrown fields, or forested wetlands.
1	= agricultural fields, athletic fields, lawns, etc.

2. Amount of Habitat

This parameter represents the amount of habitat (Types 1, 2, and/or 3) at the subject site able to support populations of *Ixodes dammini* ticks.

Point Value	Amount of Suitable Tick Habitat
5	= 80% to 100% of subject site
4	= 60% to 79% of subject site
3	= 40% to 59% of subject site
2	= 20% to 39% of subject site
1	= 0% to 19% of subject site

3. Accessibility of Habitat

This parameter provides an estimate of how accessible the suitable tick habitat at the subject site is to the human population at risk.

Point Value	Description of Accessibility
5	= suitable tick habitat is easily accessed or people are encouraged to access the subject site (picnic areas, hiking or exercise trails, campsites, place of residence, playgrounds, school yards, etc).
4	= suitable tick habitat adjacent to subject site and readily accessible.
3	= suitable tick habitat present, but access discouraged because of natural or man-made barriers (catbrier, multiflora rose, or other impenetrable vegetation; water; fences; etc.); the area is totally undeveloped; or the amount of suitable habitat is limited.
2	= access to suitable tick habitat restricted (military facilities, private clubs, etc).
1	= no access or no suitable tick habitat present.

(continued)

APPENDIX continued

4. Tick Abundance

This parameter addresses the relative number of *I. dammini* adults in habitats within the subject site. The number of *I. dammini* adults collected during 60-minute walking surveys (or extrapolated equivalent) will be used for this phase of the ecological index.

Point Value	Tick Abundance
5	= >30 *I. dammini* adults per survey
4	= 11–29 *I. dammini* adults per survey
3	= 6–10 *I. dammini* adults per survey
2	= 1–5 *I. dammini* adults per survey
1	= 0 *I. dammini* adults per survey

5. Tick Infection Rate

This parameter takes into account the percentage of *I. dammini* adults collected at the subject site that are infected with *Borrelia burgdorferi*.

Point Value	Tick Infection Rate
5	= >40% of *I. dammini* adults infected
4	= 30% to 39% of *I. dammini* adults infected
3	= 20% to 29% of *I. dammini* adults infected
2	= 10% to 19% of *I. dammini* adults infected
1	= 0% to 9% of *I. dammini* adults infected

Scoring and Response Actions—Potential Risk of Transmission

After point values are assigned to each of the three ecological parameters, the point values are totaled to achieve a final score for the subject site. The final score is then compared to the three point ranges presented below to determine the appropriate response action for the subject site.

The response action designations are to be used by the investigator to assign priorities to sites within his/her jurisdiction with respect to potential risk of Lyme disease transmission in the absence of any data on tick abundance and rates of infection by *B. burgdorferi*. This phase of the ecological index may also serve as a guide/provide justification for use of limited resources.

Scoring Range	Description of Risk	Response Action
11–15	High Risk	Place under routine tick surveillance and establish rates of infection.
6–10	Moderate [illegible]	Consider periodic tick surveillance and establishment of infection rates.
<6	Low Risk	No action necessary at this time or any time in the future.

Scoring and Response Actions—Actual Risk of Transmission

Point values relating to the habitat parameters (Parameters 1–3) are totaled first. This figure is recorded and separated from the point value for Tick Abundance (Parameter 4) by a slash (/). The point value for Tick Infection Rate (Parameter 5) is similarly separated from the remainder of the index by a slash. As such, a site receiving the maximum rating for each parameter would be recorded as 15/5/5. By recording the point values in this fashion, the ecological index becomes more descriptive, allowing the investigator to rapidly determine the relative importance of habitat, tick abundance, and tick infection rate in the assessment of the subject site.

After the point values for the five ecological parameters are recorded as above, the point values are totaled to achieve a final score for the subject site. The final score is then compared to the 5 point ranges presented below to determine the appropriate response action for the subject site.

Scoring Range	Description of Risk	Response Action
21–25	Definite Risk	Take immediate action (post area, devise control strategy).
16–20	Potential Risk	Consider action as above, but with a lower priority; place under routine tick surveillance.
11–15	Limited Risk	Perform periodic tick surveillance.
6–10	No Present Risk	Consider periodic tick surveillance; no action necessary.
<6	No Risk Likely	No action necessary at this time or at any time in the future.

Cancer Rates after the Three Mile Island Nuclear Accident and Proximity of Residence to the Plant

Maureen C. Hatch, PhD, Sylvan Wallenstein, PhD, Jan Beyea, PhD, Jeri W. Nieves, MS, and Mervyn Susser, MB, BCh

ABSTRACT

Background: In the light of a possible link between stress and cancer promotion or progression, and of previously reported distress in residents near the Three Mile Island (TMI) nuclear power plant, we attempted to evaluate the impact of the March 1979 accident on community cancer rates.

Methods: Proximity of residence to the plant, which related to distress in previous studies, was taken as a possible indicator of accident stress; the postaccident pattern in cancer rates was examined in 69 "study tracts" within a 10-mile radius of TMI, in relation to residential proximity.

Results: A modest association was found between postaccident cancer rates and proximity (OR = 1.4; 95% CI = 1.3, 1.6). After adjusting for a gradient in cancer risk prior to the accident, the odds ratio contrasting those closest to the plant with those living farther out was 1.2 (95% CI = 1.0, 1.4). A postaccident increase in cancer rates near the Three Mile Island plant was notable in 1982, persisted for another year, and then declined. Radiation emissions, as modeled mathematically, did not account for the observed increase.

Conclusion: Interpretation in terms of accident stress is limited by the lack of an individual measure of stress and by uncertainty about whether stress has a biological effect on cancer in humans. An alternative mechanism for the cancer increase near the plant is through changes in care-seeking and diagnostic practice arising from postaccident concern. (*Am J Public Health*. 1991;81:719–724)

Introduction

Shortly after the accident at the Three Mile Island (TMI) nuclear power plant on March 28, 1979, some area residents believed an increase in cancer had occurred, although a detectable excess had not been predicted. In light of the low estimates of radiation emitted, the President's Commission on the Accident at Three Mile Island concluded that the sole health consequence for the population living nearby was mental distress.[1] Because of growing interest in the effects of stress on cancer promotion and progression, we attempted an ecologic test of the relationship between cancer rates and accident stress. Natural disasters have been associated with subsequent cancer risk in previous reports,[2,3] one of which proposed that a similar study be mounted in the Three Mile Island area.[3]

At Three Mile Island, levels of distress among area residents arose immediately after the accident[4–7] and alcohol consumption rose in parallel.[8] While distress levels were elevated, they were generally in the normal range and seemed to abate within nine months, although attitudinal effects such as distrust of authorities and concerns about safety persisted.[4,5] During clean-up and krypton venting at the plant, and restart of the undamaged reactor in October 1985, turmoil at Three Mile Island was ongoing and stress and consequent distress continued or recurred. A year after the accident, a sample of community residents exhibited significantly higher levels of stress hormones and stress symptoms than residents near undamaged nuclear or coal-powered plants although, again, the levels observed were within normal limits[9]; a similar survey five years postaccident found evidence of chronic stress.[10] Periodic resurveys of other cohorts at TMI have also showed persistent distress[11] and even clinical levels of anxiety, depression and hostility, particularly in those living close to the plant.[6,12–14]

In recent years, increased attention has focused on the possibility that stress and other psychosocial factors may affect the carcinogenic process[15–21]—perhaps through the action of corticosteroids on immune function[20,21] or of stress hormones like prolactin on regulation of neoplastic cell growth.[15,19] Indirect mechanisms involving behavioral risk factors also need to be considered, as do possible interactions between stress and other risk factors. While epidemiologic studies have looked for links between psychological states, personality traits, stressful life events and the onset and development of cancer, thus far the evidence is less than conclusive.[22–38] Two recent positive reports are fairly rigorous. In one, survival time for patients with metastatic breast cancer was found to be significantly increased among those randomized to a psychosocial intervention which reduced depression, anxiety, and pain.[39] In the other, severely stressful life events proved to be strongly predictive of a first recurrence of

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Editor's Note: See also related editorial on page 687 of this issue.

breast cancer in carefully matched patients with previously operable tumors.[40]

A biologic rationale connecting stress and cancer does exist Inconsistent results from research in humans may indicate the lack of a true association, or a lack of understanding about how the neuroendocrine and immune systems mediate between psychosocial factors and cancer. In the present report, we summarize an *a priori*, if crude, test of the impact of an objective stress situation on a community's cancer rates.

Methods

The methods of this record-based geographic study are described in detail elsewhere.[41] Briefly, cancers among residents living within 10 miles of the TMI plant in the period 1975–85 were ascertained by review of patient charts at all local and regional referral hospitals (incident cases) and from vital certificates (deaths). The study area was divided into 69 small "study tracts," built up from census blocks, with populations ranging from 500-9,500 (2,300 on average). Cancers were assigned based on residence at diagnosis or at death, and for each study tract, annual rates of cancer incidence and mortality were calculated for the 11 years of the study period.

Cancer Sites Studied

In terms of the accident as a stressful event, we prespecified certain cancers or groupings of cancers for analysis, although there was little literature to guide us. Four categories were selected:

- *leukemias*, chosen because of their responsiveness to glucocorticoids, one of the "stress" hormones[42];
- *lymphomas*, which also respond to glucocorticoids and, in addition, are associated with immunosuppressive states[43];
- *"hormonally dependent" cancers* (breast, endometrium, ovary, prostate and testis), selected because stress hormones can alter concentrations of other hormones that affect cancer cells[19];
- the grouping *"all cancers"*, because in theory stress can influence the biology of all tumors through changes to the immune system.

Proximity as a Surrogate Measure of Stress

In the absence of any direct measure of stress, we used a surrogate measure supported by findings from the earlier psychosocial research at TMI. The measure is based on residential proximity to the Three Mile Island facility. A postaccident evacuation advisory issued by the Governor had focused public attention sharply on the area within a five-mile radius, with evident effect. Not only were evacuation rates higher in the five-mile radius, but so were rates of demoralization as determined by the President's Commission on Behavioral Effects.[4] Another population survey, by the Pennsylvania Department of Health,[5] found that as proximity increased so did the reported levels of perceived threat, of feeling "upset by the accident," and of psychophysiological symptoms. A study of mothers with young children found that those living in a five-mile radius of the TMI plant were more likely to experience a clinical episode of depression or anxiety than mothers living farther away.[14]

To evaluate residential proximity in the study reported here, we assigned each study tract a value corresponding to the distance between its midpoint and the TMI facility. The study area was also divided into three contiguous concentric rings based on natural breakpoints in the distribution of distances.

Proximity as Distinct from Radiation Emissions

While we treat residential proximity as a surrogate for accident-related stress, others have used it to define exposure to radiation near nuclear installations. Factors like wind direction, terrain, and elevation modify the distribution of exposures, however, and in the Three Mile Island area the terrain northeast and northwest of the plant directed emissions away from residents. In estimating radiation exposures, we developed mathematical models that take such modifying influences into account. Overlap between residential proximity and exposure to radiation emissions modeled mathematically is adjusted for in the analysis. A detailed description of the radiation emissions models is given in a separate report.[41]

Statistical Analyses

Logistic regression based on maximum likelihood estimation[44] was used to examine the age-sex-specific probability of cancer in relation to proximity of residence to the plant. Odds ratios derived from the logistic regression estimates were calculated contrasting median distance in the inner and outer rings. Indicator variables were used to adjust for a maximum of 16 age-sex categories (0–14, 15–24,. . . 65–74, 75 +). No adjustment was made for race since the population is over 96 percent White. Using aggregate data for each of the 69 study tracts, we adjusted for urbanization (population per km^2) and social class (median income; percent high school graduates), treating these measures as continuous variables.

The analyses were performed for both the preaccident period (January 1975-March 1979) and the period immediately following the accident (April 1979-December 1985). Where indicated, we used an unconditional logistic regression procedure to control for underlying heterogeneity among study tracts prior to the accident. This analysis, which is a means of adjusting for baseline risk, incorporates a parameter for each study tract, an indicator variable for period (preaccident or postaccident) and a distance variable coded zero before the accident and according to its assigned value after the accident. A conditional analysis based on a Poisson model gave similar results and is not presented here.

For descriptive purposes, we calculated standardized incidence ratios (SIRs) and standardized mortality ratios (SMRs) for the three rings at distances from the TMI facility of 0–6 km, >6–12 km, and >12 km, using age-sex-specific rates for Whites in the national Surveillance Epidemiology End Results (SEER) program (1978–81, Puerto Rico excluded).[45] However, interpretations are based on the regression analyses, which do not use an external standard.

Results

The distribution of population density, income and education by distance from the TMI plant (Table 1) shows that nearby areas are more urban and of lower socioeconomic status; these factors, as well as age and sex, are controlled in the logistic regression models.

Stress in Proximity to the Plant

For all cancers as a group, there is a statistically significant relationship between incidence rates (Table 2) after the accident and residential proximity to the plant (OR = 1.4; 95% CI = 1.3, 1.6). The association is present in both males (OR = 1.4; 95% CI = 1.2, 1.6) and females (OR = 1.5; 95% CI = 1.3, 1.7). Among the specific cancer categories tested, lymphoma shows the strongest association (OR = 1.9; 95% CI = 1.2, 3.0). Leukemias and hormonal tumors show associations of about the same magnitude as for all cancers. In addition, two common tumors

TABLE 1—Descriptive Data for Areas Defined by Distance of Midpoint from the Three Mile Island (TMI) Nuclear Plant

Distance (median) in kilometers	1980 Population[a]	Density per km^2	Median Income $	% High School Graduates
0–6 (4.3)	16,115	571	17,662	61.9
>6–12 (10.0)	60,654	316	19,452	68.8
>12 (14.2)	82,915	329	20,192	68.9

a) Midyear population estimates; adjustment has been made to the census figures for April 1, 1980.

TABLE 2—Incidence of Cancer and Proximity to TMI: Standardized Incidence Ratios (SIR)* and Odds Ratios (OR)‡ for Specific Cancer Types before and after the Accident

	Distance from Plant			
Cancer Grouping	0-6km SIR (obs)	>6-12km SIR(obs)	>12km SIR(obs)	OR (95% CI)
All Cancer				
Preaccident	0.9 (166)	0.7 (581)	0.7 (975)	1.2 (1.0,1.4)
Postaccident	1.1 (381)	1.0 (1423)	0.9 (1967)	1.4 (1.3,1.6)
Other Prespecified Sites				
Lymphoma				
Preaccident	1.7 (13)	0.7 (25)	1.0 (51)	1.2 (0.6,2.2)
Postaccident	1.5 (21)	0.9 (54)	0.9 (77)	1.9 (1.2,3.0)
Leukemia				
Preaccident	0.7 (4)	0.5 (12)	0.6 (23)	1.1 (0.4,3.0)
Postaccident	0.9 (9)	1.1 (43)	0.8 (51)	1.4 (0.8,2.5)
Hormonal[a]				
Preaccident	0.9 (45)	0.8 (165)	0.9 (298)	1.1 (0.8,1.4)
Postaccident	1.0 (86)	1.1 (408)	1.0 (581)	1.2 (1.0,1.4)
Selected Common Cancers				
Lung				
Preaccident	0.6 (17)	0.5 (61)	0.6 (116)	1.2 (0.8,1.8)
Postaccident	1.3 (67)	1.0 (214)	0.8 (282)	1.7 (1.3,2.1)
Colon				
Preaccident	0.7 (14)	0.8 (70)	0.8 (104)	1.1 (0.7,1.7)
Postaccident	1.7 (56)	1.3 (185)	1.0 (234)	1.7 (1.3,2.2)

*For calculation of SIRs, expectations are based on SEER (1978–81), age-specific rates for whites (excluding Puerto Rico). Number of observed cancers in parentheses.

‡Odds ratios comparing median distance in the inner ring with median distance in the outer ring, derived from logistic analysis adjusted for age, sex, density, median income, and education.

a) Includes breast, endometrium, ovary, prostate, and testis.

(lung and colon) also show associations with proximity to TMI (OR = 1.7 for each). The observed increase in "all cancers" thus seems to be due to increases in several cancer types.

For the "all cancer" grouping, rates in the area close to the plant are higher before the accident as well as afterwards. We corrected for the preaccident pattern by means of the procedure described above; the odds ratio adjusted for risk at baseline was reduced from 1.4 to 1.2 (95% confidence limits = 1.0, 1.4).

In Figure 1 we show incidence rates for all cancers for each year of the study period by proximity to the plant (three distance rings). By 1982, rates in the area closest to TMI were clearly elevated. The numbers of cases among residents of the inner ring rose from a yearly average of less than 50 to a high of 78 (a value that lies outside the 99% CI around 50 (33.7, 71.3), assuming the number of cases to be a Poisson variable). By 1984, the cancer rate had fallen to preaccident levels and by 1985 was lower than the rates in the more distant rings.

Mortality data (Table 3) suggest no general increase in cancer deaths with proximity to the plant in the postaccident period compared to the preaccident period. Neither do mortality rates for all cancers by year show any evidence of a change (data not presented).

Detection Bias

To test for detection bias arising from postaccident concern about cancer risk, we compared the relationship between stage at diagnosis and residential proximity to the plant in the periods before and after the event. Cancers of the breast, lung, colon, and prostate were selected since screening is common for these sites (see Table 4). With breast cancer, a shift in the postaccident period to an earlier stage at diagnosis is seen only among residents of the middle ring. For prostate cancer, residents of the inner ring actually show a decrease in the proportion of tumors diagnosed at an early stage after the accident compared to before. Lung and colon cancer show the reverse: after the accident, a higher proportion of tumors was diagnosed early among those living close to the plant.

Discussion

In an admittedly crude test of an accident-stress hypothesis, we found an increase in cancer following the accident that related to proximity of residence to the Three Mile Island nuclear plant. Cancer rates in those living nearest TMI rose in 1982, three years postaccident, remained raised for another year, and then declined. The relationship with proximity is unlikely to be explained by better case ascertainment for residents of the inner ring since, in the interests of comprehensive case-finding, we reviewed medical records at all hospitals within a 30-mile radius and at regional referral centers.

Residential proximity to the TMI plant was related to cancer rates prior to the accident as well as after, suggesting the presence of risk factors that were not sufficiently controlled by our adjustments for urbanization and social class. As a means of handling the confounding effects of unmeasured factors (e.g. cigarette smoking), we therefore adjusted for baseline risk. With this adjustment, the odds ratio for residential proximity after the accident was reduced to borderline statistical significance. Nonetheless, the postaccident pattern involving a sudden peak of excess cancers in residents near the plant does appear to be distinct from the preaccident pattern, which showed cancer rates in the inner ring to be slightly higher throughout the period.

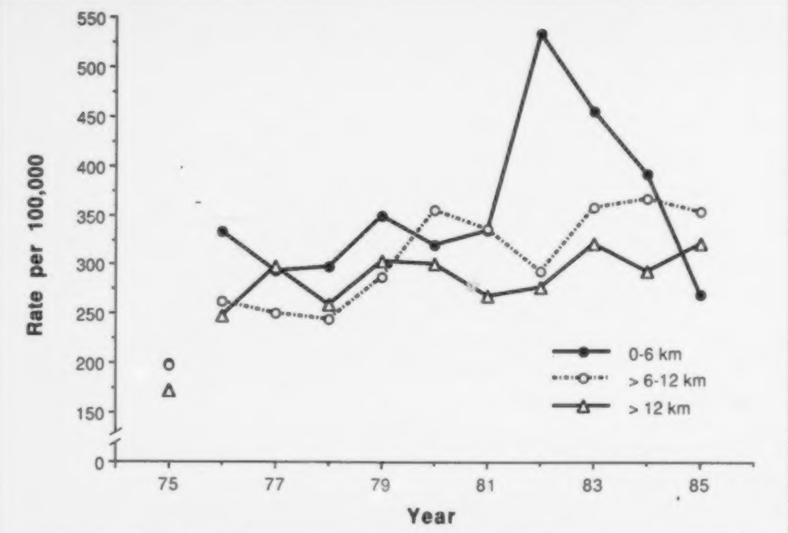

FIGURE 1—Yearly Age-adjusted Incidence Rates (100,000) of all Cancers, 1975-85, for Three Areas Defined by Distance from the TMI Nuclear Plant.

Note: In all three areas, figures were low for 1975, when hospital records were initially computerized.

TABLE 3—Mortality from Cancer and Proximity to TMI: Standarized Mortality Ratios (SMR)^a and Odds Ratios (OR)^b for Specific Cancer Types before and after the Accident

	Distance from Plant			
Cancer Grouping	0-6km SMR(obs)	>6-12km SMR(obs)	>12km SMR(obs)	OR (95% CI)
All Cancer				
Preaccident	1.3 (121)	1.0 (396)	1.0 (649)	1.2 (1.0,1.4)
Postaccident	1.0 (156)	1.1 (775)	1.0 (1119)	1.9 (0.9,1.2)
Other Prespecified Sites				
Lymphoma				
Preaccident	2.6 (9)	1.0 (15)	1.1 (28)	1.5 (0.7,3.1)
Postaccident	0.5 (3)	1.0 (27)	0.9 (38)	0.6 (0.3,1.3)
Leukemia				
Preaccident	0.7 (3)	0.6 (12)	1.1 (33)	0.5 (0.3,1.2)
Postaccident	0.8 (6)	1.2 (40)	0.9 (47)	1.0 (0.6,1.9)
Hormonal^c				
Preaccident	1.2 (21)	1.2 (86)	1.0 (118)	1.4 (0.9,2.0)
Postaccident	0.7 (19)	1.2 (155)	1.2 (229)	0.8 (0.6,1.0)
Selected Common Cancers				
Lung				
Preaccident	1.2 (28)	0.8 (80)	0.9 (135)	1.4 (1.0,2.0)
Postaccident	1.0 (40)	1.0 (164)	0.9 (241)	1.3 (1.0,1.6)
Colon				
Preaccident	0.8 (9)	0.9 (40)	1.2 (84)	0.6 (0.4,1.1)
Postaccident	1.4 (25)	1.0 (80)	1.0 (125)	1.1 (0.8,1.6)

[a] For calculation of SMRs, expectations are based on SEER (1978-81), age-specific rates for whites (excluding Puerto Rico). Number of observed cancers in parentheses.

[b] Odds ratios comparing median distance in the inner ring with median distance in the outer ring, derived from logistic analysis adjusted for age, sex, density, median income, and education.

[c] Includes breast, endometrium, ovary, prostate, and testis.

Why a peak of cancer cases should arise in 1982, three years after the accident is not at all obvious. It could relate to increased cancer surveillance near the plant, particularly as the increase appears to involve several different cancer types. If residents changed their medical care habits following the accident, so that cancer was diagnosed earlier, a "screening effect" could arise, resulting in an initial inflation in cancer rates.[46] We sought and did not find consistent evidence of earlier detection near the plant in our comparison of preaccident and postaccident diagnostic trends. The absence of an increase in mortality rates—particularly for lung cancer which is rapidly fatal—may point to a screening bias, however, as may the dip in the cancer incidence rate near the plant in 1985, the last year of the study period. Distinguishing between improved surveillance, potentially stress-related, and a direct biological effect of stress is difficult with these data, particularly if, as seems likely, stress acts to accelerate the appearance of cancer rather than to initiate new cases.

In terms of a stress hypothesis, the study's major limitation is the lack of any measure of stress at the individual level. Using distance of residence from the plant to indicate stress is bound to introduce measurement error since individuals vary in both their perception of stressful events and response to them. It does seem reasonable to assume, however, that residents living close to the plant experienced more stress on average than residents living farther away. The Governor's evacuation advisory, issued two days after the accident, focused on a five-mile radius, and investigations of possible mental health consequences found that stress responses among area residents were related to distance from the plant.[4,5,9,14] In our study, however, the proximity measure is vulnerable to migration effects: inmigrants will not have been exposed to the stress of the accident but will be considered so in the analysis, and out-migrants may be self-selected for high stress levels. Given these sources of error, if stress does underlie the observed association between proximity to the plant and cancer risk, its effect could be underestimated. On the other hand, ecological associations frequently overestimate the magnitude of effects at the individual level.[47,48] Even if the stress hypothesis holds, the mechanism could be indirect. The stress of the accident could have increased exposure to behavioral risk factors, such as cigarette smoking, but we have no data with which

TABLE 4—Distribution (%) of Selected Cancers by Stage of Diagnosis* and Proximity to the Three Mile Island Plant for the Periods before and after the Accident

Stage at Diagnosis	0-6k pre v. post		>6-12k pre v. post		>12k pre v. post	
Breast Cancer						
Local	39.3	40.4	39.4	51.8	46.0	48.5
Regional	52.6	42.5	31.3	30.7	32.2	36.5
Distant	8.2	17.0	29.1	17.8	21.7	15.0
Prostate Cancer						
Local	62.2	41.7	56.1	59.8	53.3	60.9
Regional	9.0	13.5	20.2	10.7	17.7	15.6
Distant	28.8	44.9	23.7	29.5	29.0	23.5
Lung Cancer						
Local	30.7	36.9	37.5	24.0	23.6	26.7
Regional	23.9	16.9	13.6	26.2	29.4	25.5
Distant	45.4	46.4	48.9	49.8	47.0	47.9
Colon Cancer						
Local	29.7	39.3	30.9	29.1	27.0	30.8
Regional	43.9	29.9	31.7	34.7	35.9	36.2
Distant	26.4	30.8	37.4	36.2	37.1	33.0

*Among those with known stage of diagnosis.

to evaluate this possibility. Whatever the mechanism, a true effect on cancer produced in so short a time could only manifest itself by acting on preexisting cancers.

We carried out statistical analyses to test whether the gradient in cancer risk with residential proximity—present during routine operation of the Three Mile Island plant prior to the accident, as well as after—could possibly be explained by emissions from TMI. Fitting a multivariate regression model with both measures (proximity and plant emissions modeled mathematically) actually strengthens the association of proximity with cancer incidence. Also, the pattern of rise and fall in diagnosed cancers between 1982 and 1984 would not be expected with radiation.

Conclusion

In an analysis examining cancer rates and residential proximity to the Three Mile Island nuclear plant, we observed a modest postaccident increase in cancer near TMI that is unlikely to be explained by radiation emissions. The increase resulted from a small wave of excess cancers in 1982, three years after the 1979 accident. Such a pattern might reflect the impact of accident stress on cancer progression. Our study lacked a direct, individual measure of stress, however, The most plausible alternative explanation is that improved surveillance of cancer near the TMI plant led to the observed increase. Investigations of cancer patterns in communities with future environmental incidents should consider gathering data on stress and stress-related behaviors, including change in medial care-seeking, as well as on exposure to pollutants. ☐

Acknowledgments

We are indebted to Dr. Myunghee Paik for contributions to statistical analysis, to Christine Johnsen for field work, Hung Tao for computer programming, George Colbert for cartography, and Joyce White for preparation of the manuscript. We thank the Pennsylvania State Health Data Center for providing records of cancer deaths and the 25 participating hospitals for access to records of cancer patients. The research was supported by the Three Mile Island Public Health Fund, a court-supervised fund created to address public health issues of concern to residents of the area surrounding the TMI nuclear facility; we appreciate their support.

References

1. Kemeny JG, Babbitt B, Haggerty, *et al*: Report of the President's Commission on the Accident at Three Mile Island - The Need for Change: The Legacy at TMI. ISBN 0-93578-003. Washington, DC: Govt Printing Office, 1979.
2. Bennet G: Bristol floods 1968. Controlled survey of effects on health of local community disaster. Br Med J 1970;3:454–458.
3. Janerich DT, Stark AD, Greenwald P, *et al*: Increased leukemia, lymphoma, and spontaneous abortion in western New York following a flood disaster. Public Health Rep 1981;96:350–356.
4. Dohrenwend BP, Dohrenwend BS, Fabricant JI, *et al*: Report of the Public Health and Safety Task Group on Behavioral Effects. *In*: Staff Reports to the President's Commission on the Accident at Three Mile Island Public Health and Safety Task Force #052-003-00732-1. Washington, DC: Supt of Documents, Govt Printing Office 1979; 257–308.
5. Houts PS, Miller RW, Ham KS, *et al*: Extent and duration of psychological distress of persons in the vicinity of Three Mile Island. Proc Penn Acad Sci 1980; 54:22–28.
6. Bromet E, Parkinson D, Schulberg H, *et al*: Three Mile Island: Mental Health Findings. Pittsburgh, PA: Western Psychiatric Institute and Clinic, 1980.
7. Cleary PD, Houts PS. The psychological impact of the Three Mile Island incident. J Hum Stress Spring 1984; 10:28–34.
8. Mileti DS, Hartsough DM, Madson P, *et al*: The Three Mile Island incident: A study of behavioral indicators of human stress. Int J Mass Emergencies Disasters 1984; 2:89–113.
9. Baum A, Gatchel RJ, Schaeffer MA: Emotional, behavioral and physiological effects of chronic stress at Three Mile Island. J Consult Clin Psychol 1983; 51:565–572.
10. Davidson L, Baum A: Chronic stress and posttraumatic stress disorders. J Consult Clin Psychol 1986; 54:303–308.
11. Prince-Embury S, Rooney JF: Psychological symptoms of residents in the aftermath of the Three Mile Island nuclear accident and restart. J Soc Psychol 1988; 128:779–790.
12. Bromet EJ, Parkinson DK, Schulberg HC, *et al*: Mental health of residents near the Three Mile Island reactor: A comparative study of selected groups. J Preventive Psychiatry 1982; 1:225–276.
13. Dew MA, Bromet EJ, Schulberg HC, *et al*: Mental health effects of the Three Mile Island nuclear reactor restart. Am J Psychiatry 1987; 144:1074–1077.
14. Bromet EJ, Parkinson DK, Dunn LO: Long-term mental health consequences of the accident at Three Mile Island. Int J Ment Health 1990; 19:48–60.
15. Pettingale KW: Towards a psychobiological model of cancer: Biological considerations. Soc Sci Med 1985; 20:179–187.
16. Levy SM: Behavior and Cancer - Life-style and psychosocial factors in the initiation and progression of cancer. San Francisco: Jossey-Bass, 1985.
17. Fox BH: Current theory of psychogenic effects on cancer incidence and prognosis. J. Psychosoc Oncol 1983; 1:17–31.
18. Cox T, Mackay C: Psychosocial factors and psychophysiological mechanisms in the aetiology and development of cancer. Soc Sci Med 1982; 16:381–396.
19. Lippman ME: Psychosocial factors and the hormonal regulation of tumor growth. *In*: Levy SM: Behavior and Cancer. San Francisco: Jossey-Bass 1985; 134–147.
20. Schleifer SJ, Keller SE, Stein M: Central nervous system mechanisms and immunity: Implications for tumor response. *In*: Levy SM: Behavior and Cancer. San Francisco: Jossey-Bass, 1985; 120–133.
21. Borysenko M, Borysenko J. Stress, behavior and immunity: Animal models and mediating mechanisms. Gen Hosp Psychiatry 1982; 4:59–67.
22. Shekelle RB, Raynor WJ, Ostfeld AM, *et al*: Psychological depression and 17-year risk of death from cancer. Psychosom Med 1981; 43:117–125.
23. Persky V, Kempthorne-Rawson J, Shekelle R: Personality and risk of cancer: 20-year follow-up of the Western Electric study. Psychosom Med 1987; 49:435–449.

24. Kaplan G, Reynolds P: Depression and cancer mortality and morbidity: Prospective evidence from the Alameda County study. J Behav Med 1988; 11:1–13.
25. Hahn R, Petitti D: Minnesota Multiphasic Personality Inventory-rated depression and the incidence of breast cancer. Cancer 1988; 61:845–848.
26. Zonderman AB, Costa PT, McCrae RR: Depression as a risk for cancer morbidity and mortality in a nationally representative sample. JAMA 1989; 262:1191–1195.
27. Priestman TJ, Priestman SG, Bradshaw C: Stress and breast cancer. Br J Cancer 1985; 51:493–498.
28. Greer S, Morris T: Psychological attributes of women who develop breast cancer: A controlled study. J Psychosom Res 1975; 19:147–153.
29. Horne RL, Picard RS: Psychosocial risk factors for lung cancer. Psychosom Med 1979; 41:503–514.
30. Ewertz M: Bereavement and breast cancer. Br J Cancer 1986; 53:701–703.
31. Jones DR, Goldblatt PO, Leon DA: Bereavement and cancer: Some data on deaths of spouses from the longitudinal study of Office of Population Censuses and Surveys. Br Med J 1984; 289:461–464.
32. Jones DR, Goldblatt PO: Cancer mortality following widow(er)hood: Some further results from the OPCS longitudinal study. Stress Med 1986; 2:129–140.
33. Hislop TG, Waxler NE, Coldman AJ, *et al*: The prognostic significance of psychosocial factors in women with breast cancer. J Chronis Dis 1987; 40:729–735.
34. Derogatis LR, Abeloff MD, Melisaratos N: Psychological coping mechanisms and survival time in metastatic breast cancer. JAMA 1979; 242:1504–1509.
35. Rogentine GN, van Kammen DP, Fox BH, *et al*: Psychological factors in the prognosis of malignant melanoma: A prospective study. Psychosom Med 1979; 41:647–655.
36. Greer S, Morris T, Pettingale KW: Psychological response to breast cancer: Effect on outcome. Lancet 1979; 2:785–787.
37. Pettingale K, Morris T, Greer S, *et al*: Mental attitudes to cancer: An additional prognostic factor. Lancet 1985; 1:750.
38. Cassileth BR, Lusk EJ, Miller DS, *et al*: Psychosocial correlates of survival in advanced malignant disease? N Engl J Med 1985; 312:1551–1555.
39. Spiegel D, Bloom JR, Kraemer HC, *et al*: Effect of psychosocial treatment on survival of patients with metastatic breast cancer. Lancer 1989; 1:888–891.
40. Ramirez AJ, Craig TK, Watson JP, *et al*: Stress and relapse of breast cancer. Br Med J 1989; 298:241–244.
41. Hatch M, Beyea J, Nieves J, Susser M: Cancer near the Three Mile Island nuclear plant: Radiation emissions. Am J Epidemiol 1990; 132:397–412.
42. DeVita VT, Hellman S, Rosenberg SA (eds): Cancer Principles and Practice of Oncology, 2nd Ed. Philadelphia: JP Lippincott, 1985; 1866–1897.
43. Greene MH: Non-Hodgkin's lymphoma and mycosis fungoides. *In*: Schottenfeld D, Fraumeni JF (eds): Cancer Epidemiology and Prevention. Philadelphia: WB Saunders, 1982; 754–778.
44. Breslow NE, Day NE: Statistical methods in cancer research, Vol I - The analysis of case-control studies. Lyon: IARC, 1980.
45. Young JL, Perez CL, Asire AJ, *et al*: Cancer incidence and mortality in the United States, 1973–1977, Surveillance, Epidemiology and End Results Program. National Cancer Institute monograph #57. Washington, DC: Govt Printing Office, 1981.
46. Cole P, Morrison AS: Basic issues in population screening for cancer. JNCI 1980; 64:1263–1272.
47. Piantidosi S, Byar D, Green S: The ecological fallacy. Am J Epidemiol 1988; 127:893–904.
48. Morgenstern H: Uses of ecologic analyses in epdemiologic research. Am J Public Health 1982; 72:1336–1344.

Child Care Arrangements of American Children: NCHS Report

A recent report from the National Center for Health Statistics profiles the child care arrangements received by American Children. The report, *Child Care Arrangements*, is derived from the National Health Interview Survey on Child Health, conducted by NCHS in 1988.

The survey on child care arrangements found that two thirds of all children 5 years of age and under had been in day care at some time in their lives, with the proportion increasing with age—from 56 percent for those under age 2 to 80 percent for those 4-to-5 years old. In 1988, approximately 13.3 million children were receiving child care, including 50 percent (almost 4 million) of those under the age of 2.

Of all the children receiving child care, 83 percent had mothers who were employed. The most commonly used child care arrangements were nursery schools or preschools (used by 23 percent) and care provided by a nonrelative in a home other than the child's (21 percent). About one fourth of children receiving child care were cared for alone; 35 percent were in groups of two to three children; 20 percent with four to six children; and 23 percent in groups of seven or more.

The report *Child Care Arrangements*, prepared by Deborah A. Dawson, PhD, and Virginia S. Cain, PhD, of the National Institute of Child Health and Human Development, is available from the National Center for Health Statistics, Centers for Disease Control, US Public Health Service, 6525 Belcrest Road, Hyattsville, MD 20782; (301) 436-8500.

Effectiveness of Source Documents for Identifying Fatal Occupational Injuries: A Synthesis of Studies

Nancy Stout, EdD, and Catherine Bell, BGS

ABSTRACT

Background: The complete and accurate identification of fatal occupational injuries among the US work force is an important first step in developing work injury prevention efforts. Numerous sources of information, such as death certificates, Workers' Compensation files, Occupational Safety and Health Administration (OSHA) files, medical examiner records, state health and labor department reports, and various combinations of these, have been used to identify cases of work-related fatal injuries. Recent studies have questioned the effectiveness of these sources for identifying such cases.

Methods: At least 10 studies have used multiple sources to define the universe of fatal work injuries within a state and to determine the capture rates, or proportion of the universe identified, by each source. Results of these studies, which are not all available in published literature, are summarized here in a format that allows researchers to readily compare the ascertainment capabilities of the sources.

Results: The overall average capture rates of sources were as follows: death certificates, 81%; medical examiner records, 61%; Workers' Compensation reports, 57%; and OSHA reports 32%. Variations by state and value added through the use of multiple sources are presented and discussed.

Conclusions: This meta-analysis of 10 state-based studies summarizes the effectiveness of various source documents for capturing cases of fatal occupational injuries to help researchers make informed decisions when designing occupational injury surveillance systems. (*Am J Public Health.* 1991;81:725–728)

Introduction

Identifying all of the fatal occupational injuries in the United States work force is a difficult task that has not been accomplished with precision.[1,2] Recent literature has questioned the accuracy and completeness of ascertainment of such cases by various sources of information.[1,3–6] Death certificates, Workers' Compensation files, Occupational Safety and Health Administration fatality reports, medical examiner records, reports collected by state health or state labor departments, and various combinations of these have been used to identify cases of fatal work injuries. Each of these sources was developed for different purposes, using different definitions and methods. Because there is no universally accepted gold standard against which to compare, particularly at the national level, it is difficult to measure precisely the proportion of all fatal injuries identified by a specific source.

In a smaller geographic area, such as a state, the pool of fatal work injuries can be defined as the combination of unique cases identified through several sources. Capture rates can then be estimated as the proportion of the total pool identified by each source. This method of assessing the completeness of case ascertainment by various sources of fatal work injury data has been employed in at least 10 states. Results of these studies are summarized here to provide an indication of both the overall average capture rates of the sources, and the variation in capture rates by state.

Sources of Data

The source documents used to identify fatal occupational injuries differ in their primary purposes, methods, and target populations. The advantages and limitations of various sources have been discussed elsewhere.[1,3,7] Issues of validity, completeness, accuracy, timeliness, and the usefulness of data elements in these sources are important considerations in fatality surveillance, but are outside the scope of this paper. The sources examined here are briefly described in terms of their characteristics salient to comparing capture rates.

Death certificates and medical examiner records are the only two available sources that have the potential to identify all cases of fatal work injuries in a given geographic area. Death certificates are filed for each death that occurs in the US and follow a standard format that includes an item indicating whether the death was due to an injury sustained at work. The accuracy and consistency of this item is questionable, however, as no explicit national guidelines exist that define "injury at work."[8,9] Motor vehicle crashes and homicides at work appear to be particularly underreported as work injuries on death certificates.[7,8,10,11]

In most states, medical examiner (ME) or coroner investigations are required for deaths that are due to accidents, homicides, suicides, or deaths that occur unexpectedly.[9] In jurisdictions where ME or coroner records are computerized, or at least centralized, they are a valuable source of information.[12,13] In most states,

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TABLE 1—Percent of Fatal Work Injuries Identified by Various Sources: Summary of State-Specific Studies

			Source Documents				
States	Time Period	N	% Death Certificate	% Workers' Compensation	% OSHA Fatality Reports	% Medical Examiner Records	% State Dept Health or Labor
Wisconsin[11]	1976	161	76	57	33		
Maryland[4,a]	1978	148	67	58	42	90	
Michigan[20,b]	1980	195	88				51
Alaska[21]	1980–1985	422	83	47			
Colorado[7]	1982–1984	425	86	66	32		
California[18]	1983	673	90	40	21		
New Jersey[c]	1984–1985	204	84		34	32	
Texas[d]	1986	872	67	70	27		9
Oklahoma[10]	1985–1986	329	78	53	29	82[e]	
Massachusetts[18]	10/86–11/87	86	88	62	41		22
Range (%)			67–90	40–70	21–42	32–90	9–51
Average (%)			81	57	32	68	27

[a] Susan Baker, Johns Hopkins Injury Prevention Center, personal communication, January 1991.

[b] Figures presented here exclude the "natural deaths" from the original study.

[c] Martha Stanbury, New Jersey Department of Health, personal communication, June 1990. New Jersey data exclude intentional injuries.

[d] Texas Department of Health, Occupational Safety and Health Division. Traumatic Occupational Fatalities, 1986. Unpublished report, 1989.

[e] For ME records with unclear work-relatedness, death certificates were reviewed to verify injury at work or reject false positives.

however, both ME and coroner reports exist as fragmented paper files held in various locations throughout a state, they are inconsistent among jurisdictions, and frequently do not indicate the work-relatedness of a fatal injury.[3]

Employers under Occupational Safety and Health Administration (OSHA) jurisdiction are required to report to OSHA all work-related fatalities of employees. Many of these incidents are then investigated and OSHA investigation reports are filed. Not all workers are under OSHA jurisdiction and not all incidents are investigated. Public sector employees, and self-employed workers, for example, are exempt from reporting requirements. Events such as airplane crashes, motor vehicle crashes, and homicides are investigated by other agencies, and are not routinely identified or investigated by OSHA. Recent studies also suggest substantial underreporting of fatalities that do fall under OSHA jurisdiction.[5,7,14–18]

Workers' Compensation claims can provide valuable detail about the nature of fatal injuries and accurate specification of employment. Like OSHA coverage, however, many workers are not covered by Workers' Compensation laws. Moreover, state compensation laws differ substantially so that covered worker populations are not comparable by state, and the national population of covered workers is difficult to define.

In some states, occupational fatalities are reported to the state department of labor or the state department of health. The specific types of employment and fatal events that are reported vary from state to state. For example, in Massachusetts all construction industry employers must report every accident resulting in death. The Massachusetts Department of Labor and Industries maintains records of these and other work-related fatalities reported voluntarily by employers, workers, unions, police, etc.[18]

The State Health Department source of data for the Texas study cited here refers to fatalities reported by employers participating in the Bureau of Labor Statistics (BLS) Annual Survey of Occupational Injuries and Illnesses by Industry. The survey is limited to establishments under OSHA jurisdiction, employing 11 or more workers and, as a sample survey, is not designed to capture all work fatalities.

Differences in capture rates among these sources of fatal work injury data are due in part to variation in types of employments covered by each source. Although several studies have examined capture rates of sources relative to the population covered by law to measure underreporting,[5,7,15,17] the data presented here do not address underreporting of fatal events but rather the potential of various sources to identify the universe of fatal occupational injuries.

Summary of Study Results

In each of 10 studies presented here, multiple data sources were linked, eliminating duplicate cases, to identify a total "pool" of unique fatal occupational injury cases in a state, and the proportion of that pool identified by each separate source. Table 1 summarizes the results of these studies and presents the capture rates for each source, and the range and average capture rates of the combined study results.

All 10 studies used death certificates as one source of case ascertainment and in all but two studies a greater proportion of the total pool of fatal work injuries was identified by death certificates than by any other source. The average capture rate for death certificates over all the studies was 81 percent.

Workers' Compensation files, used as a source of case ascertainment in eight states, had an average capture rate of 57 percent. The wide range (40 to 70 percent) is likely due to the different worker groups covered by the various state compensation laws.

OSHA fatality reports identified an average of 32 percent of cases. The variation in capture rates by state (21 to 42 percent) may be partially due to differences in the OSHA forms examined. In most states, first reports of fatal injuries were used to identify cases. The California[18] and New Jersey[a] studies, however, used OSHA fatality investigation reports. Since not all reported fatalities are inves-

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[a] Martha Stanbury, New Jersey Department of Health, Personal Communication, June 1990.

TABLE 2—Percent of Fatal Work Injuries Identified by Only One Source, by Source: Summary of State-Specific Studies

		Sole Source Documents				
States	Time Period	% Death Certificate	% Workers' Compensation	% OSHA Fatality Reports	% Medical Examiner Records	% State Dept. Health or Labor
Wisconsin[11]	1976	43	24	0		
Maryland[4]	1978	(N/A)	(N/A)	(N/A)	(N/A)	
Michigan[20,a]	1980	49				14
Alaska[21]	1980–85	53	17			
Colorado[7]	1982–84	34	14	0		
California[19]	1983	56	7	1		
New Jersey[b]	1984–85	31		3	12	
Texas[c]	1986	26	27	1		<1
Oklahoma[10]	1985–86	2[d]	12	<1	11[d]	
Massachusetts[18]	10/86–11/87	28	17	0		2
Range (%)		2–56	7–27	0–3	11–12	<1–14
Average (%)		36	17	<1	12	5

[a] Figures presented here exclude the "natural deaths" from the original study.

[b] Martha Stanbury, New Jersey Department of Health, personal communication, June 1990. New Jersey data exclude intentional injuries.

[c] Texas Department of Health, Occupational Safety and Health Division. Traumatic Occupational Fatalities, 1986. Unpublished report, 1989.

[d] For ME records with unclear work-relatedness, death certificates were reviewed to verify injury at work or reject false positives.

tigated, first reports are likely to identify a greater proportion of deaths than investigation reports.

Two of the three studies that included ME records identified more cases from ME records than any other source.[4,10] One study used death certificates to verify injury at work for an unknown proportion of ME records on which work relatedness was not indicated; the capture rate would have been lower if ME records had been reviewed independently.[10] In the third study, ME records identified the smallest proportion of cases.[a] This range in capture rates, from 32 percent to 90 percent illustrates the magnitude of variation by state in the potential for ME records to ascertain cases of fatal work injuries.

Occupational fatalities reported to state health departments and state departments of labor under various legislation are grouped together in Table 1 for simplicity. The average capture rate, (27 percent) is presented as a rough indication of the usefulness of such reports for identifying fatal injury cases. This average, however, is based on only three studies, and substantial differences in reporting requirements limit the comparability of these data by state.

Table 2 presents the proportion of the pool of fatal injury cases identified by only one source of information in each study. The range and average sole source capture rates provide an estimate of the proportion of the universe that might be identified by only one specific source and not the others. Obversely, the average for each source presented in this table may be interpreted as the additional proportion of the total pool of cases that might be identified by using this as an additional source of case ascertainment.

These capture rates are a function of the total pool of fatalities identified in each state, and the number and types of source documents used to define that pool. The sole source averages are presented in Table 2 as crude estimates for the purpose of comparison, rather than as precise means.

Over all the studies, death certificates uniquely identified the greatest proportion of fatal work injuries (36 percent), followed by Workers' Compensation records (17 percent), and ME records (12 percent). State labor department records (5 percent) and OSHA fatality reports (less than 1 percent) contributed relatively few additional cases to the total pool.

Discussion

Generally, if surveillance is limited to one source of information, death certificates will likely ascertain a greater proportion of the universe of fatal work injuries than will the other sources examined here. For national level surveillance, death certificates are unique among these sources in their combined potential to identify all fatal work injuries, and their comparability between states. However, the accuracy and consistency with which the injury at work items on death certificates are completed varies by state. Current efforts by the Agency for Vital Records and Health Statistics (AVRHS), National Institute for Occupational Safety and Health (NIOSH), and the National Center for Health Statistics (NCHS) to increase the validity and reliability of this item could vastly improve our ability to identify the universe of fatal occupational injuries at both state and national levels.

Under some jurisdictions, ME records may provide more complete case ascertainment than other sources. However, they also show the greatest variation by state in their ability to identify cases. Efforts to computerize ME records in a consistent format are currently underway in several states. Uniform automation of ME records could substantially improve fatal work injury surveillance within states and enable interstate comparisons.

The combined results of these studies suggest that Workers' Compensation records are not a useful sole source for case ascertainment of work fatalities. As a supplemental source, however, Workers' Compensation records could be expected to add the greatest proportion of cases to a vital records-based system. OSHA fatality reports have limited utility in fatal injury surveillance. Alone, they might be expected to identify about one-third of all cases and, as an additional source, they might capture an additional 1 percent of cases.

Because the definition of state health department and state labor department fatality reports varies so widely by state, generalized conclusions about this source cannot be drawn. In developing surveillance systems, the state-specific characteristics of this potential data source should be considered.

The combined results of these studies highlight several salient points regarding fatal work injury surveillance. The most prominent of these is that using multiple sources of data will always result in more complete case ascertainment than using one source alone.

The value added by including a subsequent source for case ascertainment depends on which source is added and its characteristics within that geographic area. For example, using state labor department records in addition to death certificates in Michigan improved case ascertainment by 14 percent.[20] In the Massachusetts study, however, where three other sources were also used and where Department of Labor reports had limited coverage, only 2 percent more cases were identified by these records.[18]

In addition to the sources discussed here, other sources should be considered to fill gaps in surveillance systems. For example, all of these sources tend to underreport occupational motor vehicle fatalities and likely undercount homicides. Police report forms and Highway Traffic Administration reports could be valuable in identifying such cases, if efforts were made to include an indication of work-relatedness on these reports.

When designing and developing fatal occupational injury surveillance efforts, cost-benefit analysis and value-added formulas are often considered to determine which sources of data will provide the most effective results given study objective and resource limitations. It is hoped that this synopsis of studies will help researchers make informed decision when designing surveillance systems, recognize limitations of various sources, and support improvements to existing sources so that we might all better understand the magnitude and nature of fatal occupational injuries in the United States. ☐

References

1. Pollack ES, Keimig DG (eds): Counting Injuries and Illnesses in the Workplace: Proposals for a Better System. Washington, DC: National Academy Press, 1987.
2. Stout-Wiegand N: Fatal occupational injuries in US industries, 1984: Comparison of two national surveillance systems. Am J Public Health 1988; 78:1215–1217.
3. Graitcer PL: The development of state and local injury surveillance systems. J Safety Res 1987; 18:191–198.
4. Baker SP, Samkoff JS, Fisher RS, Van Buren CB: Fatal occupational injuries. JAMA 1982; 248:692–697.
5. Suruda A, Emmett EA: Counting recognized occupational deaths in the United States. JOM 1988; 30:868–872.
6. Murphy DJ, Seltzer BL, Yesalis, CE: Comparison of two methodologies to measure agricultural occupational fatalities. Am J Public Health 1990; 80:198–200.
7. Colorado Department of Health: Colorado population-based occupational injury and fatality surveillance system report, 1982–1984. Denver: Health Statistics Section, Colorado Department of Health, 1988.
8. Bell CA, Stout NA, Bender TR, Conroy CS, Crouse WE, Myers JR: Fatal occupational injuries in the United States, 1980 through 1985. JAMA 1990; 236:3047–3050.
9. US Department of Health and Human Services: Medical Examiners' and Coroners' Handbook on Death Registration and Fatal Death Reporting. Hyattsville, MD: National Center for Health Statistics, 1987.
10. Russell JC: Comparison of Four Sources of Occupational Mortality Data. Doctoral Dissertation. Oklahoma City: University of Oklahoma, 1989.
11. Karlson TA, Baker SP: Fatal occupational injuries associated with motor vehicles. Proceedings of the 22nd Conference of the American Association for Automotive Medicine, Arlington Heights, Illinois, American Association for Automotive Medicine, 1978; 1:229–241.
12. Sneizek JE, Horiagon TM: Medical-examiner-reported fatal occupational injuries, North Carolina, 1978–1984. Am J Ind Med 1989; 15:669–678.
13. Centers for Disease Control: Death investigation-United States, 1987. MMWR 1989; 38:1–4.
14. Seligman PJ, Sieber WK, Pedersen DH, Sudin DS, Frazier TM: Compliance with OSHA record-keeping requirements. Am J Public Health 1988; 78:1218–1219.
15. Parkinson DK, Gauss WF, Perper JA, Elliott SA: Traumatic workplace deaths in Allegheny County, Pennsylvania, 1983 and 1984. JOM 1986; 28:100–102.
16. Stanbury M, Goldoft M, O'Leary K: Traumatic occupational fatalities in New Jersey. *In*: Pollack ES, Keimig DC (eds): Counting Injuries and Illnesses in the Workplace: Proposals for a Better System. Washington, DC: National Academy Press, 1987.
17. Stanbury M, Goldoft M: Use of OSHA inspection data for fatal occupational injury surveillance in New Jersey. Am J Public Health 1990; 80:200–202.
18. Massachusetts Department of Health: Traumatic occupational fatalities in Massachusetts, November 1986–October 1987. Boston, MA: Department of Public Health, Technical Report, March 1989.
19. Cone JE, Daponte A, Becker C, Reiter R, Balmes J: Fatal Occupational Injuries in California. Final Report MOU 86-003. Berkeley, CA: Epidemiologic Studies and Surveillance Section, California Department of Health Services, 1988.
20. Roberts C, Eyster J: A Comparison of 1980 Fatality Reports and Death Certificates in Michigan. Lansing, MI: Office of Vital and Health Statistics, Michigan Department of Public Health, 1983.
21. Schnitzer PG: The Epidemiology of Work-related Injury Fatalities in Alaska, 1980–1985. Masters Thesis. Anchorage: University of Alaska, 1987.

Female Homicides in United States Workplaces, 1980–1985

Catherine A. Bell

ABSTRACT

Background: Women, while noted for low occupational injury mortality rates, are more likely to die as victims of assault than from any other manner of injury at work.

Methods: From the National Traumatic Occupational Fatality surveillance data, 950 women were identified who were fatally assaulted at work. Homicide rates were calculated for the demographic and employment characteristics of these women. Risk ratios among types of lethal injuries were examined.

Results: During 1980–1985, the crude six-year workplace homicide rate was 4.0 deaths per million working women: one twentieth the homicide rate of the US female population. Decedents ranged from 16 years (the lowest age included in the data base) to 93 years of age. Working women older than 65 years had the highest age-specific homicide rate, 11.3 per million. Women younger than 20 had the lowest, 2.5 per million per year. Homicide rates for women of races other than White were nearly twice as high as those of Whites.

The leading causes of death were gunshot wounds (64 percent), stabbings (19 percent), asphyxiations (7 percent), and blunt force trauma (6 percent). Nearly 43 percent of the deceased women had been employed in retail trade: 8.7 per million employed women annually.

Conclusions: During 1980–1985, only 6 percent of the nation's victims of work-related injury deaths were female: 41 percent of those women were murdered. Homicide is currently the leading manner of traumatic workplace death among women in the United States. (*Am J* Public Health. 1991; 81:729–732)

Introduction

Injury surveillance conducted by the National Institute for Occupational Safety and Health (NIOSH), Division of Safety Research, has shown that from 1980 through 1985, homicide was the third leading manner of workplace death in the United States, surpassed only by motor vehicle crashes and machine-related fatal injuries. Nearly 13 percent of all occupational injury deaths were homicides. During 1980–85, 47 percent of the United States work force was female,[1] yet only 6 percent of the nearly 7,000 annual fatal work-related injuries were suffered by women: 41 percent of these women were homicide victims.[2]

Two state-based studies have shown that homicide is a prominent form of lethal occupational injury among women. In a study of fatal work-related injuries in Texas, 1975–80, 53 percent of the 348 women were murdered.[5] Only 13 percent of the fatally injured men were homicide victims.[6] The occupational homicide rate for women was one-third that for men: 7 per million employed women per year compared to 21 per million employed men. In California during 1979–81, the rate of work-related homicide among employed women was 5 per million compared to 22 per million among men.[7] Both studies showed that while a woman was at lower risk of being murdered at work than a man, a greater proportion of female traumatic occupational deaths were due to homicide.

Recently, NIOSH began using demographic and injury event information from state death certificates to examine US fatal occupation injuries, including homicides. This report describes the female victims of work-related homicide during 1980–85, as reported to the National Traumatic Occupational Fatality (NTOF) surveillance system.

Methods

The NTOF data base is composed of death certificates from all state vital statistics reporting units in the US that meet the following criteria: 1) age at death—16 years or older; 2) a positive response to the "Injury at Work" item; and 3) an external cause of death (International Classification of Diseases, Ninth Revision, E800-E999). The methods and limitations that pertain to NTOF injury surveillance are described fully elsewhere.[8]

All NTOF records of female deaths from 1980 through 1985 that reported homicide as the manner of death were selected for this analysis. Homicide, death due to injuries purposely inflicted by another person, was identified using the element in Part II of the death certificate that queries "Accident, Suicide, Homicide, or Pending" because not all NTOF records had coded cause of death information at the beginning of the study. No effort was made to exclude deaths due to legal intervention. Recording practices in Louisiana, Nebraska, New York, and Oklahoma prevented identification of work-related

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From the National Institute for Occupational Safety and Health in Morgantown, WV, where the author was with the Division of Safety Research at the time of the study. Address reprint requests to Chief, Injury Surveillance Section, Division of Safety Research, National Institute for Occupational Safety and Health, S-109, 944 Chestnut Ridge Road, Morgantown, WV 26505. This paper, submitted to the Journal February 20, 1990, was revised and accepted for publication February 19, 1991.

TABLE 1—Female Occupational Homicides in the United States[a] by Year, 1980–85: Numbers and Annual Rates[b]

Year	Number	Deaths per 1,000,000
1980	176	4.75
1981	163	4.36
1982	171	4.44
1983	143	3.65
1984	147	3.59
1985	150	3.57
All Years	950	4.04

[a] Excluding deaths from Louisiana, Nebraska, New York, and Oklahoma.
[b] Deaths per 1,000,000 workers.

TABLE 2—Female Occupational Homicides in the United States[a] by Age, 1980–85: Numbers and Six-year Rates[b]

Age (years)	Number	Deaths per 1,000,000
16–19	43	2.50
20–24	155	4.47
25–34	286	4.34
35–44	159	3.17
45–54	138	3.81
55–64	101	4.11
65+	68	11.28

[a] Excluding deaths from Louisiana, Nebraska, New York, and Oklahoma.
[b] Deaths per 1,000,000 workers.

intentional injuries over the study period, so homicide data from these four states were not available.

Employment information was coded from the "usual" industry and occupation entries on the certificates. The Standard Industrial Classification[9] system was used to categorize usual industry of employment. Occupation information was coded into major groups with the 1970 Bureau of Census Classifications for deaths in 1980 through 1982 and the 1980 Bureau of Census Classifications for deaths in 1983 through 1985. The two coding schemes ensured agreement between incidence and underlying employment information because the source of the population data used in the rate calculations changed coding schemes in 1983.[10,11]

To calculate mortality rates, the Annual Summaries of EMPLOYMENT AND EARNINGS[12] provided denominator data for the US. The GEOGRAPHIC PROFILE OF EMPLOYMENT AND UNEMPLOYMENT[13] was used to correct the denominator data used in age-, race-, and employment-specific rates by excluding the estimated work force of the four states which did not provide homicide data to NTOF (nearly 10 percent of the female work force of the US).

Homicide frequencies and annual and six-year mortality rates were calculated for demographic and employment characteristics. Victims whose occupations or industries of employment were unclassifiable were not included in employment-specific rate calculations, although they were included in all other rates. Age- and race-specific rates were estimated. Race-specific rates were calculated for Whites (including Hispanics), Blacks, and Others (Asians and American Indians). Definitive employment data were not available for Hispanics, Asians, or American Indians. Risk ratios (RR) contrasting types of injury (e.g. firearms versus not firearm) within racial subsets were estimated.

Deaths assumed to involve close physical contact, such as stabbing or beating, were compared to those resulting from gunshot wounds that could be inflicted from a distance. Two categories of close physical contact deaths were defined: multiple stabbing and a broader category that included all rape, stabbing, slashing, blunt force, and manual asphyxiation deaths. These were contrasted to the gunshot wounds through risk ratio estimates. Ninety-five percent Taylor Series confidence intervals were constructed around all risk ratio estimates.[14]

Results

NTOF data described 950 women who were murdered at work during 1980–85 (Table 1). The women's ages ranged from 16, the youngest documented in NTOF data, to 93 years. The six-year age-adjusted homicide rate was 4.5 deaths per million working women. The highest homicide rate, 11.3 per million, was among working women 65 years of age and older (Table 2).

Black women were 1.8 times more likely to be killed than Whites. The RR for homicide among Blacks contrasted to all other women was 1.7 (95% CI: 1.4, 2.0) and for the Other racial category was 1.9 (95 % CI: 1.4, 2.5). Hour of injury was reported for 680 cases: homicides were most frequent between 4:00 and 5:00 pm. However, 30 percent of the women were murdered between 6:00 pm and midnight and 69 percent died between 3:00 pm and 7:00 am.

Industry of Employment

Nearly 43 percent of the homicide victims were employed in the Retail Trade Industry which also had the highest six-year homicide rate, 8.7 per million retail employees. Forty-one percent of these women worked in food stores, and 34 percent in eating and drinking establishments.

The Transportation, Communication, and Public Utilities Industry had the second highest rate, 4.0 per million: 34 percent were employed in motor freight transportation and warehousing and 18 percent in local passenger transportation with buses and taxis. While 23 percent of the women worked in the Service Industry, their homicide rate was quite low, 2.22 per million: 22 percent of these women worked in education, 16 percent in health services, and 13 percent in hotels, motels, and other lodging.

In Public Administration, 33 percent of the women were employed in justice, public order, and safety, 28 percent in general government, and 23 percent in the military. Industry of employment was not coded for 14 percent of the decedents, due to lack of specific employment information on the death certificates (Table 3).

Occupation

Despite the changes in the occupation classification schemes in 1983, 71 percent of the murdered women were employed in only four occupational groups: sales personnel (n = 184), clericals (n = 169), service employees (n = 182), and supervisors (executives, managers, and administrators) (n = 151). During 1980-82, retail store clerks and cashiers, classified as clericals, accounted for 26 percent of the decedents' occupations: their homicide rate was only 3.4 per million. Sales workers, such as sales clerks and owners of retail establishments, died at a rate of 8.3 per million. During the next three years, retail clerks and cashiers were classified as sales occupations. These accounted for 27 percent of the 1983–85 deaths with a three-year rate of 7.6 per million.

For the six-year period, 105 of the 175 women in service occupations were employed in eating and drinking establishments. At least 52 of these worked in bars, taverns, or lounges. Nineteen women were employed as law enforcement officers or security guards. Lack of national

TABLE 3—Female Occupational Homicides in the United States[a] by Industry, 1980–85: Numbers and Six-year Rates[b]

Industry	Number	Deaths per 1,000,000
Agriculture/Forestry/Fishing	4	1.11
Construction	6	2.15
Manufacturing	44	1.23
Transportation/Communication/Public Utilities	38	3.97
Wholesale Trade	9	1.52
Retail Trade	405	8.66
Finance/Insurance/Real Estate	55	2.78
Services	219	2.20
Public Administration	40	3.92
Not Classified	130	—

[a]Excluding deaths from Louisiana, Nebraska, New York, and Oklahoma.
[b]Deaths per 1,000,000 workers.

estimates for employment in these specific occupations made rate calculations impossible.

Employment as a supervisor increased a woman's risk of homicide. Decedents most commonly managed restaurants, apartment complexes, or retail establishments. Ten percent of the women owned their own businesses.

Region

Only 24 (3 percent) of the women were murdered outside the states where they resided. Despite the lack of reporting from Louisiana and Oklahoma, over half (52 percent) of the murders took place in the South at a rate of 5.7 per million women working in the reporting states. The Western states had the second highest rate, 4.1 per million, and 23 percent of the homicides. The North Central states, where Nebraska deaths were not reported, had a homicide rate of 2.5 per million and 18 percent of the deaths. Only 7 percent of the fatalities were reported from the Northeast at a rate of 1.2 per million. This region's figures are seriously compromised by the omission of New York deaths from the data. Of the 46 states reporting, rates indicated that women were at highest risk of being murdered at work in Nevada, Mississippi, the District of Columbia, Florida, and Texas.

Method of Homicide

Firearm injuries caused 609 deaths (64 percent): 148 (16 percent) noted multiple gunshot wounds. Stabbing and slashing caused 181 deaths (19 percent). Multiple stabbing was mentioned in 103 cases (11 percent). Decedents were asphyxiated in 69 (7 percent) or died from blunt force injuries in 57 (6 percent) of the cases. Together, fires, explosions, motor vehicle impacts, poisoning, and sexual assault were reported in fewer than 4 percent of all cases.

Lethal gunshot wounds were most frequent in the 25–54 year age range. The firearm homicide rate among Black females averaged 4.4 per million compared to 2.2 per million among Whites. Black women were slightly more likely to die from gunshot than other wounds (RR, 1.1, 95% CI: 1.0, 1.2). The risk of Hispanic women dying from firearm relative to other injuries (RR) was 1.4 (95% CI: 1.2, 1.6).

Multiple stabbings were prominent, but not significantly elevated, among the oldest women. However, the risk of an elderly woman dying from any form of physical contact relative to gunshot wounds (RR) was 3.6 (95% CI: 2.6, 4.9). Asian and American Indian women were slightly more likely to be killed in trauma involving physical contact than from gunshot wounds (RR of 1.4, 95% CI: 1.0, 2.0), while Black women were more likely to die from gunshot wounds (RR = 0.7, 95% CI: 0.5, 0.9).

Discussion

While the limitations of death certificate information in general[15–18] and NTOF in particular[8] have been well defined, a few merit discussion in terms of occupational homicide surveillance. Surveillance based on death certificates alone underreports cases of fatal occupational injuries. State studies have shown between 67 percent and 88 percent of all traumatic workplace deaths can be identified using death certificates alone.[19] Legal sources such as police files and medical examiner records are more detailed; however, they do not regularly identify work-related cases and are not readily accessible in most US jurisdictions. Death certificates provide systematic accounts of each traumatic death in the US, and not those reported to have occurred at work. Death certificates record "usual" occupation and industry of employment, not necessarily employment at the time of injury. Studies show that between 64 percent and 74 percent of reported usual occupations correspond with decedents' most recent occupations, and between 60 percent and 76 percent of the usual industry entries correspond with most recent industries of employment.[16-18]

Both formal state policies and individual practices affect case ascertainment and data accuracy. The most notable weakness is lack of standard criteria for completing the injury at work item; it is not rigorously defined and is subject to interpretation. For example, in Louisiana a state policy existed where "injury" was interpreted as "accidental injury." Until 1985, the injury at work item was not completed for intentional injuries. Even when injuries that take place at work are correctly recorded, some states automate that information only for unintentional injuries. In these states all homicides and suicides must be screened to find each noted as having occurred at work. This screening is tedious and through human error intentional injury cases may be missed.

A certifier may also impose an idiosyncratic definition of work that excludes or obscures employment. No prostitutes or illicit drug dealers are identifiable in the NTOF data. Juveniles are rarely considered "at work." Deaths perpetrated by acquaintances or family members may be screened by a certifier who considers "at work" to mean solely work-related. Homicides are also misclassified as accidents, suicides, or deaths of undetermined cause. For such reasons, the cases here underreport victimization among working women in the US.

While such factors can hinder hazard identification and ultimately injury control, NTOF surveillance rapidly identified homicide as the major occupational hazard for the nation's women. Homicide risk may be associated with exposure to the public, the exchange of money, and evening employment.[5–7,20] Women employed in the retail industry, particularly in food stores, were at greatest risk. Many owned or managed groceries, taverns, and restaurants. Elevated homicide rates in motor freight and warehousing, local passenger transportation, government, and law enforcement call for heightened safety for workers in these jobs. Detailed studies of homicide risk in education, health services, hotels, motels, and other lodging places are also warranted.

The role of victim age in these deaths is still not clear. Older women may be attractive targets and/or less likely to survive traumatic assault. This analysis also showed that older women were often self-employed or small-business owners. While Davis, *et al*, argued that high workplace homicide rates in Texas women age 65 years or older did not result from underenumerated employment,[5] the person-year denominators used in the NTOF age rates may distort the risk estimates. For example, low rates among young women may reflect part-time work. Age-specific homicide rates should be examined using hour-based employment information when available.

Most of the women died from gunshot wounds. Close physical contact deaths were significantly elevated among older White women and among Asians, a group generally noted for low risk of violent death.[21] Because control strategies may depend on discrimination between robbery assault and personal or sexual violence, more detailed studies are needed on the circumstances of workplace homicides.

At least one major convenience store chain has implemented robbery deterrence programs which couple behavioral and environmental components.[22] In evaluated programs, employees have been trained in techniques of conflict resolution and nonviolent response.[21–24] Locked drop-safes, well-lit parking lots, and a work area openly visible to the public are environmental components that appear to reduce robbery and associated mortality.[22]

Convenience stores are essentially small businesses with control strategies that might prove useful in small, owner-operated businesses. Before recommending use of their control strategies developed in other environments, performance in the new environment should be rigorously evaluated.

Hazardous industries may also benefit from regulation, particularly where employers have not reduced homicide risk. The Occupational Safety and Health Administration (OSHA) standards do not yet explicitly address safety from workplace violence, but an increasing number of local ordinances mandate hours for store operation or the presence of multiple employees. The next step in reducing the risk of workplace homicide is to evaluate the effectiveness in various work settings of existing as well as new control strategies. ☐

References

1. US Department of Labor, Bureau of Labor Statistics: Geographic Profile of Employment and Unemployment. Labor force status of the civilian noninstitutional population 16 years and over by state, sex, age, race, and marital status, annual averages. Bulletin 3311 (1986).
2. Centers for Disease Control: Occupational homicides among women — United States, 1980–1985. MMWR 1990; 39:544–522.
3. Baker SP, Samkoff JS, Fisher RS, VanBuren CB: Fatal occupational injuries. JAMA 1982; 248:692–697.
4. Centers for Disease Control: Fatal occupational injuries—Texas, 1982. MMWR 1985; 34:130–139.
5. Davis H, Honchar PA, Suarez L: Fatal occupational injuries of women, Texas 1975–1984. Am J Public Health 1987; 77:1524–1527.
6. Davis H: Workplace homicides of Texas males. Am J Public Health 1987; 77:1290–1293.
7. Kraus JF: Homicide while at work: Persons, industries, and occupations at high risk. Am J Public Health 1987; 77:1285–1289.
8. Bell CA, Stout NA, Bender TR, Conroy CS, Crouse WE, Myers JR: Fatal occupational injuries in the United States, 1980 through 1985. JAMA 1990; 263:3047–3050.
9. Executive Office of the President, Office of Management and Budget: Standard Industrial Classification Manual, 1972. Washington, DC: Govt Printing Office, 1972.
10. US Department of Labor, Bureau of Labor Statistics: Employment and Earnings. Household data annual averages. 31, 1984.
11. US Department of Commerce: 1980 Census of Population, Alphabetic Index of Industries and Occupations, 2nd Ed. Washington, DC: Department of Commerce, May 1981.
12. US Department of Labor, Bureau of Labor Statistics: Employment and Earnings. Household data annual averages. 28(1), 1981; 29(1), 1982; 30(1), 1983; 31(1), 1984; 32(1), 1985; 33(1), 1986.
13. US Department of Labor, Bureau of Labor Statistics: Geographic Profile of Employment and Unemployment. Labor force status of the civilian noninstitutional population 16 years and over by state, sex, age, race, and marital status, annual averages. Bulletins 2111 (1982), 2156 (1982), 2170 (1983), 2216 (1984), 2234 (1985) 3311, (1986).
14. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research: Principles and Quantitative Methods. Belmont, CA: Lifetime Learning Publications, 1982.
15. Kirchner T, Nelson T, Burdo H: The autopsy as a measure of accuracy of the death certificates. N Eng J Med 1985; 313:1263–1269.
16. Schade WJ, Swanson GM: Comparison of death certificate occupation and industry data with lifetime occupational histories obtained by interview: Variations in the accuracy of death certificate entries. Am J Ind Med 1988; 14:121–136.
17. Illis WR, Swanson GM, Satariano ER, Schwartz AG: Summary measures of occupational history: A comparison of latest occupation and industry with usual occupation and industry. Am J Public Health 1987; 77:1532–1534.
18. Davis H: The accuracy of industry data from death certificates for workplace homicide victims. Am J Public Health 1988; 78:1579–1581.
19. Stout NA, Bell CA: Effectiveness of source documents for identifying fatal occupational injuries. Am J Public Health 1991; 81:725–728.
20. Hales T, Seligman PD, Newman SC, Timbrook CL: Occupational injuries due to violence. JOM 1988; 30:483–487.
21. Centers for Disease Control: Homicide Surveillance: High Risk Racial and Ethnic Groups: Blacks and Hispanics, 1970 to 1983. Atlanta: CDC, November 1986.
22. Crow W, Bull JL: Robbery deterrence: An applied behavioral science demonstration. La Jolla, CA: Western Behavioral Sciences Institute, 1975.
23. Felson RB, Steadman HJ: Situational factors in disputes leading to criminal violence. Criminology 1983; 21:59–74.
24. Erickson R, Crow W: Violence in business settings. Am Behav Scientist 1980; 23:717–743.

Occupation, Industry, and Fatal Motor Vehicle Crashes in 20 States, 1986–1987

Dana P. Loomis, MSPH, PhD

ABSTRACT

Background: Motor vehicles are a leading cause of injury on and off the job.

Methods: To describe the occurrence of fatal motor vehicle crashes in the working-age population, a case-control study was conducted among persons 15–64 years old who died in 1986 or 1987 in any of 20 states reporting death certificate occupational data to the National Center for Health Statistics. Cases were occupants of motor vehicles (excluding motorcycles) who died of injuries sustained in a crash (E810-E825, 9th revision International Classification of Diseases), and controls died of any other cause.

Results: Transportation-related occupations had more than the expected number of deaths for men (odds ratio = 1.6, 95% confidence interval = 1.4 – 1.9) and women (odds ratio = 2.1, 95% confidence interval = 1.0 – 4.5), as did managerial occupations (odds ratio = 1.3 and 1.7, 95% confidence interval = 1.1 – 1.5 and 1.3 – 2.2 for men and women, respectively). Men in mining and oil drilling occupations and the wholesale trade industry also had excess crash deaths. Among women, additional excesses were observed among professional specialists and in several groups with few deaths, including agriculture, construction, and the military, whereas homemakers had markedly fewer vehicle crash deaths than expected.

Conclusions: These findings encourage further investigation of this important safety hazard, but also call attention to a need for more detailed studies and improved data. (*Am J Public Health.* 1991;81:733–735)

Introduction

Motor vehicles cause more fatal occupational injuries in the United States than any other agent.[1] Previous studies providing data on this problem have focussed on single states or selected occupational groups,[2–6] but this investigation, using death certificate data from the National Center for Health Statistics, was undertaken to provide a description of the overall epidemiological pattern of motor vehicle-related fatalities in the United States working population to be used in planning future research and prevention efforts.

Methods

The subjects of this study were residents of 20 states (Alaska, Colorado, Georgia, Indiana, Kansas, Kentucky, Maine, Missouri, Nevada, New Hampshire, New Mexico, North Carolina, Ohio, Oklahoma, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and Wisconsin) who died in 1986 or 1987. Only these states coded occupation and industry data from death certificates and reported them to the National Center for Health Statistics (NCHS) in at least one of those two years. Occupation and industry coding are done by the states according to the 1980 US Census Bureau occupation and industry classification system.[7]

Subjects were selected from 1986 and 1987 mortality data tapes from NCHS. All 5,031 men and 1,776 women 15–64 years old who died of injuries received as drivers or passengers in crashes of motor vehicles other than motorcycles (coded E810-E825 with 4th digit equal to 0 or 1 according to the International Classification of Diseases, 9th Revision), and who had an occupation other than "student," "volunteer," "never worked," or "retired" on the death certificate were selected as cases. A control group of 7,167 men and 4,369 women was formed by selecting a 5 percent simple random sample of deaths from all causes other than motor vehicle crashes from the same NCHS tapes using restrictions identical to those applied to the cases.

Standard case-control odds ratios were computed by treating each occupational category in succession as the "exposed" group, and all other categories combined as "unexposed."[8] The odds ratios can be interpreted as the ratio of the observed number of motor vehicle crash fatalities in each occupational category to that expected based on the experience of all others.[9] Age-adjusted odds ratios were estimated by the Mantel-Haenszel method, and test-based confidence intervals were computed for both the crude and adjusted estimates.[8] Because female homemakers were the largest single occupational group, they were considered separately using all women with employment outside the home as the unexposed referent, but were excluded from all other analyses involving women.

Results

Occupation

Crude and age-adjusted odds ratios for motor vehicle crash fatalities by occupation are shown in Table 1. Without adjustment for age, men in white collar oc-

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TABLE 1—Odds Ratios (95% confidence intervals in parentheses) for 1986–87 Motor Vehicle Crash Deaths for Men and Women Aged 15–64 years by US Census Bureau Occupational Category (unexposed referent group is all other occupations)

	Men				Women[a]			
Occupation	Cases	Controls	Crude	Age-adjusted	Cases	Controls	Crude	Age-adjusted
Professional specialists	243	501	0.7 (0.6, 0.8)	0.9 (0.8, 1.1)	175	310	0.9 (0.8, 1.1)	1.2 (1.0, 1.5)
Technicians & related support workers	99	104	1.4 (1.0, 1.8)	1.1 (0.8, 1.5)	58	74	1.3 (0.9, 1.9)	1.2 (0.8, 1.8)
Sales	403	605	0.9 (0.8, 1.1)	1.2 (1.0, 1.4)	164	246	1.0 (0.8, 1.3)	0.9 (0.7, 1.2)
Administrative support & clerical	158	255	0.9 (0.7, 1.1)	0.9 (0.7, 1.1)	269	503	0.9 (0.7, 1.0)	0.8 (0.6, 0.9)
Services	369	597	0.9 (0.8, 1.0)	0.7 (0.6, 0.8)	314	592	0.9 (0.7, 1.0)	0.8 (0.6, 1.0)
Farming & agriculture occupations	238	391	0.9 (0.7, 1.0)	1.0 (0.8, 1.2)	19	27	1.2 (0.7, 2.1)	1.4 (0.6, 3.0)
Forestry, fishing & hunting occupations	48	54	1.3 (0.9, 1.9)	1.0 (0.7, 1.6)	0	1	0 —	0 —
Mechanics & repairers	351	422	1.2 (1.0, 1.4)	1.1 (0.9, 1.4)	6	10	1.0 (0.4, 2.7)	0.5 (0.1, 1.8)
Construction trades	571	701	1.2 (1.1, 1.3)	1.0 (0.9, 1.2)	9	2	7.5 (2.0, 27.6)	5.7 (1.2, 26.7)
Extractive occupations	56	58	1.4 (1.0, 2.0)	1.8 (1.2, 2.8)	1	1	1.7 (0.1, 25.8)	1.0 (0.0, 855.0)
Precision production workers	260	452	0.8 (0.7, 1.0)	1.0 (0.9, 1.3)	27	54	0.8 (0.5, 1.3)	1.0 (0.5, 1.8)
Machine operators & tenders	334	503	0.9 (0.8, 1.1)	0.9 (0.7, 1.0)	125	195	1.1 (0.9, 1.4)	1.1 (0.8, 1.5)
Fabricators, assemblers & hand workers	146	174	1.2 (1.0, 1.5)	1.0 (0.8, 1.4)	26	55	0.8 (0.5, 1.3)	0.7 (0.4, 1.4)
Inspectors, testers, samplers & weighers	30	40	1.1 (0.7, 1.7)	1.4 (0.7, 2.6)	15	27	0.9 (0.5, 1.7)	1.5 (0.8, 2.8)
Transportation & material moving occupations	537	658	1.2 (1.1, 1.3)	1.6 (1.4, 1.9)	19	16	2.0 (1.0, 3.8)	2.1 (1.0, 4.5)
Handlers, cleaners, helpers & laborers	683	826	1.2 (1.1, 1.3)	0.7 (0.6, 0.8)	80	113	1.2 (0.9, 1.6)	1.0 (0.7, 1.4)
Military occupations	142	203	1.0 (0.8, 1.2)	0.9 (0.7, 1.3)	10	2	8.3 (2.3, 29.7)	1.5 (0.2, 13.4)
Homemakers	4	5	1.1 (0.3, 4.2)	0.3 (0.0, 1.5)	316	1460	0.3 (0.2, 0.3)	0.4 (0.4, 0.5)

[a] Among women the referent group for each occupation in the paid workforce is all other such occupations, i.e. homemakers are excluded; for female homemakers, the referent group is all occupations except homemaker.

TABLE 2—Odds Ratios (95% confidence intervals in parentheses) for 1986–87 Motor Vehicle Crash Deaths for Men and Women Aged 15–64 years by US Census Bureau Industry Category (unexposed referent group is all other industries)

	Men				Women[a]			
Industry	Cases	Controls	Crude	Age-adjusted	Cases	Controls	Crude	Age-adjusted
Mining	96	107	1.3 (1.0, 1.7)	1.3 (0.9, 1.9)	1	4	0.4 (0.1, 3.5)	0.1 (0.0, 1.1)
Construction	919	1075	1.3 (1.2, 1.4)	1.0 (0.9, 1.1)	29	16	3.0 (1.7, 5.5)	3.2 (1.4, 7.4)
Manufacturing	1249	1880	0.9 (0.9, 1.0)	1.0 (0.9, 1.1)	303	482	1.1 (0.9, 1.2)	1.1 (0.9, 1.4)
Transportation, communication & utilities	499	705	1.0 (0.9, 1.1)	1.4 (1.2, 1.6)	74	76	1.7 (1.2, 2.3)	2.0 (1.3, 3.0)
Wholesale trade	165	197	1.2 (1.0, 1.5)	1.4 (1.1, 1.8)	24	40	1.0 (0.6, 1.7)	0.8 (0.4, 1.5)
Retail trade	603	685	1.3 (1.2, 1.5)	1.0 (0.8, 1.1)	340	437	1.4 (1.2, 1.6)	0.9 (0.7, 1.1)
Finance, insurance & real estate	107	206	0.7 (0.6, 0.9)	1.0 (0.8, 1.3)	92	128	1.2 (0.9, 1.6)	1.2 (0.8, 1.7)
Business & repair services	320	391	1.2 (1.0, 1.4)	1.0 (0.8, 1.2)	38	55	1.2 (0.8, 1.8)	0.9 (0.5, 1.6)
Personal services	80	149	0.8 (0.6, 1.0)	0.7 (0.5, 0.9)	93	262	0.6 (0.4, 0.7)	0.7 (0.5, 1.0)
Entertainment & recreation services	46	68	1.0 (0.7, 1.4)	0.7 (0.5, 1.2)	14	22	1.1 (0.5, 2.1)	1.1 (0.4, 2.9)
Professional & related services	249	534	0.7 (0.6, 0.8)	0.9 (0.7, 1.0)	338	673	0.8 (0.7, 0.9)	0.9 (0.8, 1.1)
Public administration	180	347	0.7 (0.6, 0.9)	1.0 (0.8, 1.3)	49	138	0.6 (0.4, 0.8)	0.6 (0.4, 1.0)
Military	147	209	1.0 (0.8, 1.2)	1.0 (0.7, 1.3)	9	2	7.5 (2.0, 27.6)	1.3 (0.1, 12.5)

[a] Excludes homemakers.

cupations, except for technicians and related workers, tended to have essentially the expected number of crash deaths or somewhat fewer, whereas most blue collar occupations had a small excess of deaths. With age adjustment, "extractive" occupations (miners and oil well drillers), and transportation and material moving occupations had the highest odds ratios, with 1.8 and 1.6 times more deaths than expected, respectively. Executives, administrators, managers, and sales workers had more modest excesses of deaths.

For women employed outside the home, some of the largest excesses of vehicle crash fatalities occurred in occupations with relatively few deaths, notably construction trades, transportation, agriculture, and the military. Odds ratios were also elevated, although to a generally lesser degree, in more populous categories, including managerial occupations, professional specialists, and technicians. Female homemakers had very low crash mortality compared to women with paid employment.

Industry

Odds ratios indicating the association of motor vehicle crash deaths with Census Bureau industry categories are shown in Table 2. Among men, most industries had essentially the expected number of crash deaths. However, the categories of transportation, communication, and utilities and wholesale trade had 40 percent more deaths than expected when adjusted for age.

Construction, followed by transportation, communication, and utilities, were the industrial categories with the largest excesses of crash fatalities among women employed outside the home, with about three and two times the expected number of cases, respectively. The more modest excess of deaths among women in the finance, insurance, and real estate category may be more quantitatively important given the larger number of deaths in that group. Public administration and personal services are populous categories with substantially fewer deaths than expected. Crude and adjusted odds ratios tended to be similar except for the military category, for which the odds ratio declined sharply with adjustment for age.

Discussion

This 20-state mortality study describes patterns of motor vehicle crash fatality for men and women by occupation and industry. Such information has not been available before for a geographically diverse population including a wide spectrum of occupations. The vital statistics data used in this study offer several advantages for identifying groups of workers at increased risk: death registration is complete, comparability of data between cases and noncases is assured, and large studies including many states and all occupational groups can be done at minimal cost. On the other hand, studies of this type share generic limitations of design and data quality which have been discussed elsewhere.[9–11]

The major interpretational challenge of this study is that the data do not allow injuries which occurred while the decedent was working to be distinguished from those which occurred during other activities. To minimize the proportion of nonworkers in the study, only individuals ages 15 to 64 years with an identifiable occupation indicated on their death certificate were included. However, deaths due to injuries which did not occur at work were also necessarily included, because the states do not report to NCHS the death certificate field that identifies on-the-job injuries.[10] A field coded for the place of injury (for example, industrial place, home, or farm) is available from NCHS public data, but it does not appear to effectively distinguish occupational injuries.[12]

Although it would be helpful to separate injuries which occur on the job from those taking place elsewhere, the ability to do so is not central to all potential uses of these data. The present analysis based on occupational groups also approximates the socioeconomic pattern of motor vehicle crash mortality. In addition, the data are relevant to injury prevention efforts, because some programs directed toward workers, like the Occupational Safety and Health Administration's proposed workplace driver training program,[13] have the potential to reduce injury risk both on and off the job. Accurate identification of mortality excesses due to on-the-job hazards remains important for more specific interventions, such as equipment modifications, however.

In spite of some disadvantages, the data used in this study appear to be sensitive enough to capture previously-observed excess vehicle crash mortality among transportation workers.[2,6] This should lend some confidence to the interpretation of the occupational patterns of mortality which emerge from these data. Comparison of the crude and age-adjusted odds ratios may also be useful for interpreting the variation in mortality among different groups of workers: the adjusted odds ratios should most accurately reflect the inherent hazardousness of a job, while the crude measures are also sensitive to the sociodemographic composition of the workforce.

Because of the large number of injuries and deaths due to the use of motor vehicles in work, these hazards should be a priority for injury research and prevention. This study suggests that further investigation of managers and other seemingly low-risk groups, in addition to transportation workers, might be warranted. However, the availability of appropriate data remains an obstacle. Uniform coding of the death certificate indication of injury at work and its inclusion in NCHS public mortality data would improve future surveys based on those data, but studies using more detailed information on individuals and vehicles used for work are also needed to identify factors which could be modified to prevent motor vehicle injuries to workers. ☐

Acknowledgments

I thank Carol Runyan and Thomas Cole for their constructive criticism of an early version of this paper.

References

1. National Safety Council: Accident Facts: 1989 Ed. Chicago, IL: National Safety Council, 1989; 33.
2. Baker SP, Samkoff JS, Fisher RS, Van Buren CB: Fatal occupational injuries. JAMA 1982; 248:692–697.
3. Centers for Disease Control: Fatal occupational injuries—Texas, 1982. MMWR 1985; 34:130–139.
4. Sniezek JE, Horiagon TM: Medical-examiner-reported fatal occupational injuries, North Carolina, 1978–1984. Am J Ind Med 1989; 15:669–678.
5. Robinson CC, Kuller LH, Perper J: An epidemiologic study of sudden death at work in an industrial county. Am J Epidemiol 1988; 128:806–820.
6. Marine W, Garrett C, Keefer S, Vancil R, Hoffman R, McKenzie L: Occupational injury deaths in Colorado 1982–1987. Denver, CO: Colorado Department of Health, 1990.
7. US Bureau of the Census: Alphabetical Index of Industries and Occupations. US Dept of Commerce PHC80-R3. Washington, DC: Govt Printing Office, 1982.
8. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research: Principles and Quantitative Methods. Belmont, CA: Lifetime Learning Publications, 1982; 343–352.
9. Miettinen OS, Wang JD: An alternative to the proportionate mortality ratio. Am J Epidemiol 1981; 114:144–148.
10. Bell CA, Stout NA, Bender TR, Conroy CS, Crouse WE, Myers JR: Fatal occupational injuries in the United States, 1980 through 1985. JAMA 1990; 263:3047–3050.
11. Dubrow R, Sestito JP, Lalich NR, Burnett CA, Salg JA: Death certificate-based occupational mortality surveillance in the United States. Am J Ind Med 1987; 11:329–342.
12. Trent RB: Locations of fatal work injuries in the United States: 1980 to 1985. JOM 1989; 31:674–676.
13. US Department of Labor, Occupational Safety and Health Administration: Occupant protection in motor vehicles. Federal Register 1990; 55:28728–28740.

An Analysis of Occupational Blood Lead Trends in Manitoba, 1979 Through 1987

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ABSTRACT

Background: While regulations for workplace lead exposure become more strict, their effectiveness in decreasing blood lead concentrations and the method by which this is attained have not been evaluated.

Methods: An analysis was conducted of 10,190 blood lead samples from employees of 10 high-risk workplaces collected in Manitoba, 1979–87, as part of regulated occupational surveillance.

Results: A significant decrease in blood lead concentrations was observed overall as well as for each individual company. A 1979 government regulation to reduce blood lead to below 3.38 μmol/L (70 μg/dl) was followed by a drop in blood lead concentrations; a 1983 order to reduce blood leads to below 2.90 μmol/L (60 μg/dl) was not followed by such a drop. Longitudinal analysis by individual workers suggested that companies were complying by use of administrative control, *i.e.*, removing workers to lower lead areas until blood lead levels had fallen, then returning them to high lead areas.

Conclusion: Focusing upon blood lead as the sole criterion for compliance is not effective; regulations must specifically require environmental monitoring and controls. Biological surveillance serves as "back-up" to environmental surveillance and this database illustrates the usefulness of a comprehensive centralized surveillance system. (*Am J Public Health*. 1991;81:736–740)

Introduction

Although lead poisoning has been documented since antiquity, occupational lead intoxication continues to occur.[1-3] Moreover, there is growing consensus that lead causes toxic injury to humans at levels of exposure that only a decade ago were thought to be safe. As Landrigan notes,[1] these subclinical changes represent truly harmful outcomes and are not merely homeostatic or physiologic "adjustments" to the presence of lead.

The US Occupational Safety and Health Administration (OSHA) standard, since 1978, has required the removal from exposure of workers who have confirmed blood lead levels of 2.9 μmol/L (60 μg/dl) or higher, an average of 2.4 μmol/L (50 μg/dl) or higher on three tests, or a medical condition contraindicating continued exposure to lead. Once removed, workers cannot return to a job where they will be exposed to lead until the blood lead level is 1.93 μmol/L (40 μg/dl) or less or the employee no longer has a medical contraindication to lead exposure.[2] Environmental monitoring is also required, to ensure a permissible exposure limit of 50 μg/m.[3]

Evidence is lacking as to the effectiveness of any regulation with respect to lead poisoning. While some states maintain heavy metal registries, these only required the reporting of blood lead concentrations above 1.93 μmol/L (40 μg/dl) (until recently when some states dropped their required reporting level to 1.21 μmol/L [25 μg/dl]). Hence, longitudinal follow up of individuals, as well as by workplace, is not possible.

Manitoba, a Canadian province with a population of one million, has a list of high-risk workplaces that are under government surveillance, as well as a centralized provincial laboratory where all occupational blood lead samples are analyzed. With over 11,000 blood lead concentrations recorded in this data base for more than a decade, linked by individual as well as by workplace, it is possible to evaluate trends in the province overall, as well as by individual company. It is also possible to assess concurrent turnover or rotating of staff (administrative controls). Utilizing this blood lead data base, we address specifically the following questions:

- What is the extent of occupational lead intoxication?
- Have mean blood lead concentrations decreased following government improvement orders and tighter standards?
- What has been the role of administrative measures and engineering controls in any such decrease?

Methods

Lead Measurements

A total of 10,190 blood lead tests were performed as a result of regulatory sur-

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veillance reported from 10 high-risk workplaces. Five workplaces had regular medical monitoring programs throughout the period 1979–87 inclusive, and five conducted comprehensive and continuous programs for at least two years during this period. In other workplaces the medical monitoring was sporadic and voluntary. All blood lead analyses were conducted at the provincial government's Technical Services Laboratory located in Winnipeg, Manitoba. The laboratory is accredited by the American Industrial Hygiene Association and participates in the Blood Lead Proficiency Testing program of the Centers for Disease Control in Atlanta, as well as the program conducted by the Centre de Toxicologie in Quebec. Blood leads are analyzed using the Fernandez Method.[4]

Those whose blood lead concentrations exceeded 2.41 μmol/L (50 μg/dl) on at least one occasion, had an annual frequency of testing ranging from one to 21, median of four and mode of four, whereas those whose blood lead concentrations were always less than 1.93 μmol/L (40 μg/dl) had an annual average testing frequency ranging from one to nine, with median of one and mode of one. To adjust for this variation, a quarterly median was taken for each individual. If an individual had only one blood lead test in a given year, the same result was entered in all four quarters for that year. If an individual had blood tests monthly, only the median of each quarter was entered for analysis.

As measures of blood lead concentrations tend to be skewed, most of the analyses were smoothed (using a median quarterly or annually as the case may be) and the overall medians analyzed. Contingency table analysis allowed the comparison of aggregated distributions of blood lead concentrations, while linear regression analysis assessed company trends over the nine-year period, and Mantel-Haenszel trend tests evaluated specific target proportions over shorter time segments (i.e. proportion of values over 70 μg/dl during the three years following 1979 and proportion over 60 μg/dl in the three years following 1983).

To gain insight into the mechanism underlying the longitudinal variations of individual blood lead concentrations, first order auto correlations to assess the influence of one result on the next among individual workers and testing the run of signs of first differences for randomness to assess individual patterns were performed.[5,6] The overall effect of administrative controls as hypothesized by a negative association between last blood lead concentration nearing the target level and current blood concentrations was tested using the Mantel-Haenszel chi-square stratified by regulatory period.

Industrial hygiene observations and measurements were gleaned from the records of the provincial government's workplace safety and health inspectorate.

Occupational Lead Exposure and Regulatory Process

Workplace safety and health is a provincial responsibility in Canada, with each province enacting its own legislation and regulations. The Workplace Safety and Health Act in Manitoba, proclaimed in 1977, empowers the minister to issue an order requiring an occupational health service at designated workplaces, with penalty of fine for noncompliance. This permitted the minister in charge of occupational health for the province to issue an order to five companies in 1979 to reduce blood lead concentrations to below 3.86 μmol/L, (80 μg/dl). There was no mention of environmental monitoring in the order. An individual was to be removed from the plant when his blood lead concentration exceeded 3.86 μmol/L or transferred to a low lead exposure area when the blood lead ranged from 3.38–3.86 μmol/L (70–80 μg/dl). In 1983, after considerable debate, the 1979 order was revised reducing the blood concentration removal level to 3.35 μmol/L (70 μg/dl), with workers to be transferred to low lead areas when the blood concentration ranged from 2.90–3.38 μmol/L (60–70 μg/dl). There was no mention of differential levels for women, or of environmental monitoring.

The five firms that received this order consisted of one battery manufacturer, two primary lead-using industries, one secondary lead smelter, and one combined battery breaking/secondary smelter operation. These companies all operated continuously from 1979 to 1987 with the exception of the combined battery breaking/secondary smelter operation which was shut down briefly for economic reasons during 1983. Three other companies conducted biological monitoring over part of the same period; a primary copper smelter with additional production of zinc and lead concentrate (1982–87), a battery breaking operation in a reclamation yard (1985–87), and an aluminum forging operation (1985–86) with incidental lead exposure.

In 1986 following years of controversy and no written policy, the Workers' Compensation Board (WCB) in Manitoba essentially established a blood lead concentration of 2.41 μmol/L (50 μg/dl) as a compensable level.[7] Sixty-three claims for lead poisoning had been allowed by the Manitoba WCB prior to 1986; 54 were allowed in the two years following this policy statement.

Results

Table 1 contains the 10,190 blood lead concentrations of employees who worked in the 10 designated plants between 1979 and 1987 inclusive. The proportion of values over 3.86 μmol/L (80 μg/dl) has steadily decreased; there is a statistically significant difference in the distribution of lead concentrations over the years, ($p < .0001$). There were no essential changes in this distribution when one entry per quarter for each worker is included regardless of the number of tests performed on that worker during that particular year (data available from author on request). Figure 1 illustrates the trends in median blood lead concentrations over time in each of the five companies that operated throughout this period (with the exception of a brief plant closure in 1983 in one). Median blood lead levels by year and type of plant are given in the Appendix.

A separate analysis was conducted to assess the proportion of blood lead concentrations over 3.38 μmol/L (70 μg/dl) in the three years following the 1979 order which required a reduction to below this level. Both the overall trend as well as the trend within each of the five firms subject to this order showed a highly significant decrease in the proportion of blood leads over this level (overall from 7.9 percent in 1979 to 0.8 percent in 1982). When a similar assessment was done for the 1983 order, only one of the then six companies showed a statistically significant decrease in proportion of blood leads over 2.90 μmol/L (60 μg/dl), the new target level (data available from author on request). Two companies, a secondary smelter and a combined battery breaking/secondary smelter operation, which had 11.2 percent and 11.8 percent of blood levels over 2.90 μmol/L (60 μg/dl), respectively, at the time of the 1983 order, had 13.9 percent and 16.9 percent over 2.90 μmol/L (60 μg/dl), respectively, three years later.

Industrial hygiene investigation revealed that after the first government intervention, some hygiene and/or engineering controls were implemented in all of the firms in question with each firm adopting a unique strategy depending on its condi-

TABLE 1—Percentage Distributions of 10,190 Blood Lead Concentrations in Manitoba 1979–87 Inclusive

Blood Lead Concentration*	1979	1980	1981	1982	1983	1984	1985	1986	1987
<20	11.5	9.0	8.6	21.2	38.5	36.7	16.4	26.2	37.7
20–29	12.1	15.8	20.4	24.0	22.4	21.8	20.5	18.7	14.7
30–39	13.9	16.8	18.5	19.4	14.2	17.0	19.6	16.4	13.1
40–49	15.4	19.4	17.8	14.2	14.5	14.2	20.7	16.3	15.3
50–59	16.5	18.1	14.5	11.9	6.8	7.2	15.0	15.2	10.5
60–69	12.8	9.6	9.2	6.7	2.7	2.2	5.7	5.0	6.8
70–79	8.4	7.0	7.2	1.9	0.8	0.3	1.1	1.4	1.4
≥80	9.4	4.3	3.8	0.7	0.1	0.6	1.0	0.8	0.4
Total Samples	962	1268	1103	699	973	877	1460	1699	1149

*µg/dL. Conversion factor to SI Units (µmol/L) = .04826

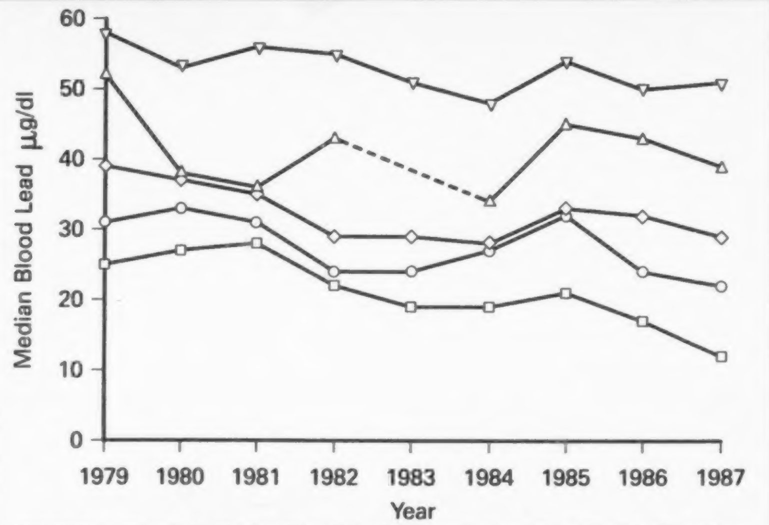

FIGURE 1—Trends in Median Blood Lead Concentrations Over Time in Five Companies Operating during 1979–87

tion. Thereafter, companies seemed to be making greater use of administrative controls, i.e. removing workers to lower lead-in-air areas when blood lead concentrations approached the action level, only to return them to the higher areas after their blood lead concentration had fallen. Industrial hygiene inspections found for example, that while the lead-in-air levels had dropped from 300 µg/M^3 to less than 150 µg/M^3 in a primary lead using company, between 1979 and 1983, such a decrease was not observed in the inspection of a secondary lead smelter where environmental levels continued to exceed 300 µgm/M^3. A second secondary smelter demonstrated a reduction of near 150 µg/M^3 on average but some areas, notably the furnace area, demonstrated no substantial reduction in air-borne lead with concentrations approaching 400 µg/M^3 and personal protection being used as a protective strategy. No ongoing environmental or personal monitoring programs were in place and government monitoring was generally complaint-based. Environmental data are thus incomplete.

To further investigate the apparent failure of the 1983 order to produce a continuing decrease in blood lead concentrations, longitudinal analysis was performed for all workers who ever had values of 2.90 µmol/L (60 µg/dl) or greater. A graph for these workers individually showed a marked "titration" pattern, indicating use of administrative controls. Figure 2 shows two typical examples of this pattern. It demonstrates that as a worker's blood lead concentration approaches the action levels he is removed to a lower lead area, and after his blood lead concentration falls, he is probably returned to a higher lead-in-air concentration since his blood lead level rises. This titration pattern in individual workers was present throughout the period of data collection. Its existence was further demonstrated by the significant ($p = 0.0001$) finding that if a worker's blood lead was less than 2.41 µmol/L (50 µg/dl) his next blood lead would be higher and vice versa. This indicates that an overall decrease in lead exposure in the plant was not occurring because the lead exposure was being averaged over a pool of employees. Four of the eight companies still in operation in 1985 had 18–50 percent of blood lead concentrations over 2.90 µmol/L (60 µg/dl); one other company brought its percentage down to 1.9 percent, while the remaining three companies had had less than 1 percent even prior to the 1983 order. The workforce turnover rates were higher in the four companies with poor compliance than in the four with lower proportion of concentrations over 2.90 µmol/L (60 µg/dl) (data available from author on request). There was much less evidence of titration in the four with better compliance (data available from author on request).

Discussion

The prevention of lead poisoning requires: 1) knowledge of dose response relationships with establishment of an acceptable target blood lead "threshold" to reduce the potential for subclinical lead toxicity, 2) effective surveillance of the environment and of the at-risk population, and 3) action to reduce lead exposure at the source. Considerable knowledge exists with respect to "threshold" levels of exposure for different adverse effects, as summarized elsewhere.[2] There is a need to intensify the monitoring of lead levels in the at-risk population in order to take action which would result in a safer workplace. Methods used to monitor trends of occupational illness and injury[8–13] are usu-

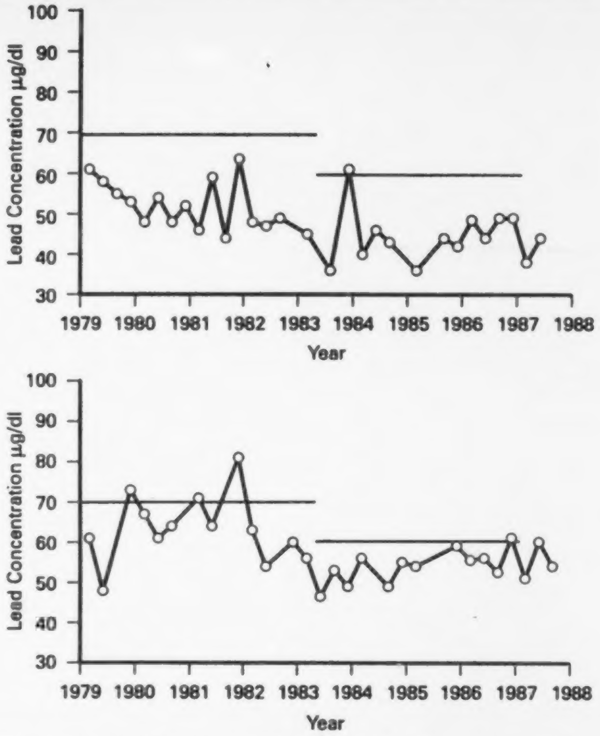

FIGURE 2—Blood Lead Concentrations in Two Individual Workers 1979–87

ally developed primarily for other purposes, making them less useful for occupational health surveillance. Most states have reporting systems specifically for occupational disease,[9] but the completeness of reporting is rarely satisfactory due to a lack of effective centralized monitoring, and disincentives for physicians and others to report occupational disease.[8,9]

One of the main purposes of surveillance systems is to evaluate the effectiveness of worksite control programs.[11] However, there is little information addressing the effectiveness of government regulatory strategy in controlling work-related illness and injury, and there has certainly been controversy[14] in this regard. This study illustrates an overall decrease in the extent of lead poisoning over the last decade. However, on careful examination it is apparent that while the 1979 government order to reduce blood lead concentrations to below 3.38 µmol/L (70 µg/dl) was effective, the 1983 order to reduce blood leads to below 2.90 µmol/L (60 µg/dl) was less so. Thus the conclusion regarding the effectiveness of Manitoba's regulatory strategy is mixed.

Our findings show that when biological monitoring is the only criterion for compliance, utilization of administrative controls enables companies to comply with stricter regulations without decreasing overall lead exposure. The practice of administrative controls is problematic for several reasons. It results in subjecting more people to lead exposure, thereby failing to reduce overall lead exposure; it often causes employment disruption with its consequent socioeconomic consequences; it usually fails to reduce individual body burden of lead. The oscillations observed below the action level (Figure 2) do not represent lead exposure followed by lead excretion. Rather, due to man's relative inability to excrete lead, the downstrokes represent lead storage within the body. The end result of this process is chronic lead poisoning. This finding is supported by the clinical observations of the clinician authors (AY and MT) in which modest blood lead elevations in several long-term employees have been noted to be accompanied by marked increases in erythrocyte protoporphyrin concentrations.

In 1987 the minister responsible for occupational health in Manitoba issued a further order requiring "... a worker to be removed from the lead containing environment until his/her lead level returns to acceptable levels below 2.41 µmol/L (50 µg/dl). The worker may work in a job with minimal lead exposure (30 µg/M^3) considered as an 8 hour average ..." Moreover, the order stated that the exposure to the worker shall be "no greater than 50 µg/M^3 ... Only upon a detailed engineering assessment by a qualified engineer can consideration be given to permit exposure levels ... to exceed 50 µg/M^3." As most Canadian jurisdictions utilize the American Conference of Governmental Industrial Hygienist (ACGIH) level of 150 µg/M^3, this order was considered quite progressive by some and draconian by others. Regardless of the appropriateness of the lead-in-air concentration chosen, the data presented here demonstrate that the inclusion of an environmental target level was a needed addition. Nonetheless, biological monitoring must continue, with the data base described above serving an important role in monitoring the effectiveness of this new regulation.

In conclusion, while appreciation of lead as a subclinical toxin is essential, and a comprehensive blood lead surveillance system for high risk industries is crucial, unless biological monitoring is in conjunction with environment monitoring and measures to decrease exposure at the source, the overall goal of reducing occupational lead poisoning will not be achieved. □

Acknowledgments

This research was funded in part by the University of Manitoba Research Development Fund.

We acknowledge with thanks the cooperation of Will Grieve and staff of the William M. Ward Technical Services Laboratory, Department of Environment, Manitoba.

References

1. Landrigan PJ: Toxicity of lead at low dose. Br J Ind Med 1989; 46:593–596.
2. Goldman RH, Baker EL, Hannan M, Kamerow DB: Lead poisoning in automobile radiator mechanics. N Engl J Med 1987; 317:214–218.
3. Cullen MR, Robins JM, Eskenazi B: Adult inorganic lead intoxication: Presentation of 31 new cases and review of recent advances in the literature. Medicine 1983; 62:221–247.
4. Fernandez FJ: Micromethod for lead determination in whole blood by atomic absorption with use of the graphite furnace. Clin Chem 1975; 21:558–561.
5. Edgington ES: Probability table for number of runs of signs of first differences in ordered series. J Am Stat Assoc 1961; 56:156–159.
6. Gibbons JD: Nonparametric Statistical Inference. New York: McGraw Hill, 1971; 62–66.

7. Workers' Compensation Board (WCB) Manitoba. Board Order No. 198/86. Criteria for Acceptability of Claims for Lead Toxicity. Manitoba: WCB, 1986.
8. Baker EL, Melius JM, Millar JD: Surveillance of occupational illness and injury in the United States: Current perspectives and future directions. J Public Health Policy 1988; 9:198–221.
9. Muldoon JT, Wintermeyer LA, Eure JA, *et al:* Occupational disease surveillance data sources, 1985. Am J Public Health 1987; 77:1006–1008.
10. Seligman PJ, Halperin WE, Mullan RJ, Frazier TM: Occupational lead poisoning in Ohio: Surveillance using workers' compensation data. Am J Public Health 1986; 76:1299–1302.
11. Baker EL, Honchar PA, Fine LJ: Surveillance in occupational illness and injury: Concepts and content. Am J Public Health 1989; 79(Suppl):9–11.
12. Melius JM, Sestito JP, Seligman PJ: Occupational disease surveillance with existing data sources. Am J Public Health 1989; 79(Suppl):46–52.
13. Blanc PD, Rempel D, Maizlish N, Hiat P, Olson KR: Occupational illness: Case detection by poison control surveillance. Ann Intern Med 1989; 111:238–244.
14. Mendeloff JM: The Dilemma of Toxic Substance Regulation: How Overregulation Causes Undergraduation. Cambridge, MA: MIT Press, 1988.

APPENDIX—Median Blood Lead vs Year for Lead Exposed Workers from Five Manitoba Companies

	1979–82				1983–87				1979–87
	Observations		P values		Observations		P values		
Firm	Mean	Range	Trend Test[a]	Linear Regression	Mean	Range	Trend Test[b]	Linear Regression	Linear Regression
★ = Secondary smelter	104	80–136	.022	.7706	96	80–108	.364	.1998	.321
△ = Combined battery breaking and secondary smelter[c]	268	128–50	.000	.0001	95	56–148	.618	.0537	.4100
◇ = Battery manufacturer	446	420–452	.005	.0001	526	484–572	.038	.0018	.0265
○ = Primary lead user	174	140–212	N/A	.0001	132	32–168	N/A	.211	.049
□ = Primary lead user	688	504–860	.000	.0001	480	464–504	.471	.1438	.0004

[a] Trend Test for Proportions >70 μg/dl
[b] Trend Test for Proportions >60 μg/dl
[c] Data unavailable for 1983 due to plant closure

Occupational Carpal Tunnel Syndrome in Washington State, 1984–1988

Gary M. Franklin, MD, MPH, Joanna Haug, MSc, Nicholas Heyer, PhD, Harvey Checkoway, PhD, and Neil Peck, MPA

ABSTRACT

Background: There are no published population-based studies of occupational carpal tunnel syndrome (OCTS) using a strict case definition. Most studies are either industry specific or present patient self-report of symptoms.

Methods: We conducted a population-based incidence study of OCTS using the Washington State Workers' Compensation database. Incident OCTS claims were identified with paid bills for physician reported ICD codes 354.0 and 354.1.

Results: There were 7,926 incident OCTS claims identified for the years 1984–1988, which yields an industry-wide incidence rate of 1.74 claims /1,000 FTEs. The mean age (37.4 years) and female/male ratio (1.2:1) in this population differ from those reported in nonoccupational carpal tunnel studies (mean age, 51 years; female/male ratio, 3:1). The female-specific OCTS incidence rate increased significantly during the study period. The highest industry specific OCTS rates were found in the food processing, carpentry, egg production, wood products, and logging industries.

Conclusion: Demographic differences and industry-specific rates consistent with workplace exposures suggest that OCTS is distinct from CTS occurring in nonoccupational settings. Workers' compensation data proved useful in identifying high risk industries. (*Am J Public Health.* 1991;81:741–746)

Introduction

Occupational carpal tunnel syndrome (OCTS) is a major cause of lost work days and workers' compensation costs in the United States. A recent Centers for Disease Control report, based on physician reporting, estimated that 47 percent of carpal tunnel cases are work-related.[1] Industry-specific prevalence rates of OCTS have been reported to be as high as 15 percent.[2] Most such reports reflect prevalence rates in individual plants or small populations of workers.

Population-based data on the incidence of OCTS has been difficult to generate. The principal problem appears to be that OCTS does not have a unique code in the US Department of Labor Z–16.2 coding system. All wrist disorders are aggregated, and specifically diagnosed cases of OCTS cannot be identified routinely. A recent report using Wisconsin workers' compensation data could identify only "potential" OCTS cases.[3]

Washington State Department of Labor and Industries' claims and medical bill payment information systems were utilized to generate age-, sex- and industry-specific OCTS rates for the years 1984–88. In the context of this study, the validity of using such an administrative data base in performing epidemiologic research was investigated.

Methods

Workers' Compensation System

The Washington State Department of Labor and Industries (L&I) is the sole regulator of workers' compensation coverage in Washington State covering approximately 1.3 million full-time workers in 1988. Coverage for roughly one-third of these workers is through self-insurance administered company programs, and the other two-thirds of workers are covered by the Washington State Fund.

Claims Management Data Base

The L&I claims management data base consists of two major data processing systems. The Medical Information and Payment System receives all billing information generated by provider medical bills. This system records such relevant items as dates of service, all associated procedure (CPT) codes, and physician diagnosis by International Classification of Disease (ICD)[4] code for each provider visit.

The Labor and Industries Industrial Insurance System contains all data necessary for administration of claims. Recorded data include age, sex, claim ID, claimant ID, social security number, nature of injury (Z16.2) codes, time loss, and medical aid paid-to-date. In addition, industry type is coded using the Washington Industrial Classification (WIC) system.

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TABLE I—NIOSH Case Definition for Occupational Carpal Tunnel Syndrome (CTS)

A. One or more of the following *symptoms* affecting at least part of the median nerve distribution: paresthesias, hypoesthesia, pain or numbness; and
B. *Objective findings* consistent with CTS:
   Either
   1) *Positive physical examination* findings −(+) Tinel's or Phalen's or decreased or absent sensation to pin prick in median nerve distribution of the hand; or
   2) *Nerve conduction findings* indicative of median nerve dysfunction across the carpal tunnel; and
C. Evidence of work-relatedness—a history of a job involving one or more of the following activities *prior to* development of CTS symptoms:
   1) *Frequent, repetitive* use of the hand or wrist,
   2) Regular tasks requiring the generation of *high force* by the hand,
   3) Regular or sustained tasks requiring *awkward hand positions,*
   4) Regular use of *vibrating hand tools,*
   5) Frequent or prolonged *pressure over the wrist or base of the palm.*

Source = Adapted from Cummings et al.[1]

Incident OCTS Claim Definition

A data extract was generated of all medical bill line items with an ICD code of 354.0 (carpal tunnel syndrome) or 354.1 (median nerve neuritis) from both the claims and medical bill payment systems. The data set included both compensable (time loss) and non-compensable claims for which medical bills were paid between January 1, 1984 and December 31, 1988. Self-insured claims were excluded from both the numerator and denominator in subsequent analyses due to incomplete data.

An incident claim was defined as the first appearance of a paid bill for a claimant with a physician ICD diagnosis of 354.0 or 354.1 between 1984–88. An algorithm was developed to identify unique claimants by claimant ID and social security numbers. This process ensured the removal of multiple claims for the same condition or conditions recurring at a later date, and eliminated problems of missing claimant identifiers for archived claims.

Denominators

OCTS incidence rates by year and industry were estimated directly using worker hours reported to L&I. Worker hours were converted to full time equivalent workers (FTEs) by FTEs = total hours reported/2000.

Age- and sex-specific denominators were not available from worker hours reported to L&I and were estimated by using other available data. Census data for Washington State (1980) were used to estimate an age/sex distribution for the employed population. The Office of Financial Management state and county projected population figures for 1980–91 were used to determine percent changes in the age/sex-specific strata between 1980 and the years of interest.

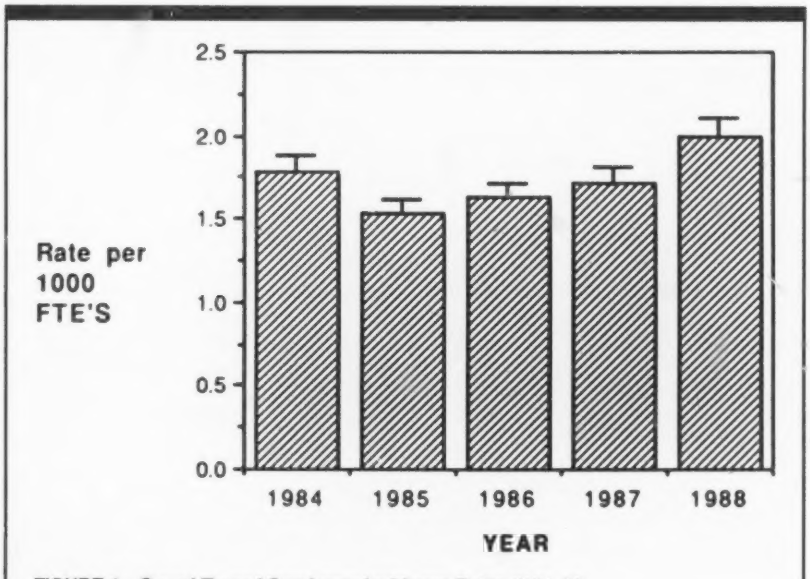

FIGURE 1—Carpal Tunnel Syndrome Incidence Rates 1984–88

Industry-Specific OCTS Rates

Worker hours reported to L&I represent hours of exposure by Washington Industrial Classification (WIC). An industrial classification is a grouping of industries that share similar workplace exposures. Currently there are 975 industrial classifications. OCTS rates are presented using WIC classifications as a surrogate for industry type. In order to eliminate unstable rates, only those classifications with a minimum of 25 OCTS cases over the five-year study period were included in the study.

Misclassification Studies

Two separate sub-studies were performed in an attempt to define potential misclassification: 1) a validation study using the medical records for a random sample of 245 incident claims during the period 1984–88 to estimate (a) the proportion of claims with a documented physician diagnosis, and (b) the proportion of claims that fit the clinical criteria for case definition according to National Institute for Occupational Safety and Health (NIOSH) standards (Table 1)[1], and 2) a review of ICD coding errors associated with a separate sample of L&I claimants who received carpal tunnel surgery between 1984–87.

Statistical Methods

Incidence rates by age, sex, year, and industry are expressed as numbers of OCTS claims/1,000 FTEs. A statistical comparison of rates for male/female differences and test for trend over time were done using formulas reflecting a Poisson distribution.[5,6] Each industry-specific rate was compared to the industry-wide rate, and a crude incident rate ratio with a 95 percent confidence interval was developed.[6]

Results

OCTS Incidence by Age, Sex, and Year

Figure 1 shows that the incidence rate for OCTS claims increased from 1.78/1,000 FTEs in 1984 to 2.00/1,000 FTEs in

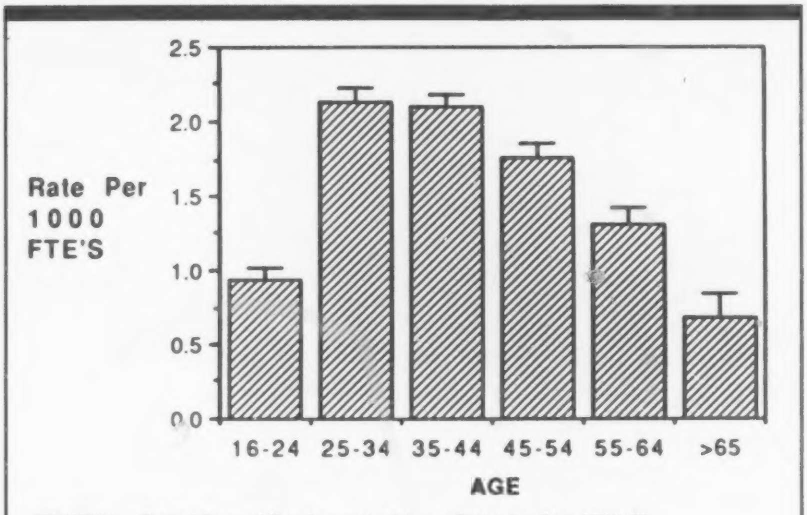

FIGURE 2—Carpal Tunnel Syndrome Incidence Rates by Age 1984–88

1988 (1,533 claims in 1984; 1,320 claims in 1985; 1,475 claims in 1986; 1,642 claims in 1987; and 1,956 claims in 1988). The trend of increase is statistically significant ($p < .01$).

The OCTS incidence rate peaked in the 25–44 age group (mean age 37.4 years) (Figure 2). The overall incidence rate for females (1.96/1,000 FTEs) was significantly greater than that for males (1.58/1,000 FTEs, $p < 0.001$), and this difference has increased over time (Figure 3). The female OCTS incidence rate increased significantly between 1984–88 ($p < 0.005$), while the male incidence rate remained stable with no statistically significant trend.

Industry-Specific OCTS Incidence Rates

For the period 1984–88, the industry-wide OCTS incidence rate was 1.74 claims/1,000 FTEs. The top 20 industries are presented sorted by rate (Table 2) and by total number of incident claims (Table 3). The highest OCTS rate of 25.7 claims/1,000 FTEs for oyster, crab, and clam packing was approximately 15-fold higher than the industry-wide rate (1.74 claims/1,000 FTEs).

Misclassification

OCTS Case-Definition Study—In order to validate the methodology used to identify OCTS claims, medical records for a random sample of 245 incident OCTS claims were reviewed. Two hundred and one (201) out of 245 claims (82 percent) did have a physician diagnosis of OCTS documented in the medical records. The proportion of incident claims without a clear diagnosis of OCTS was consistent across age, sex, and year; no significant differences in proportions were seen within these categories. A high correlation was found between physician diagnosis of OCTS and the criteria set out by NIOSH for case definition. Of the 201 cases with a physician diagnosis in the medical records, 186 (93 percent) met NIOSH standards for OCTS.

An attempt was made to reduce the potential misclassification due to "false" OCTS claims (missing a clear physician diagnosis) by testing more restrictive incident claim definitions (Table 4). The baseline inclusion criteria of at least one visit with ICD codes 354.0 or 354.1 resulted in a "false" claims rate of 18 percent (44/245) in the validation study. Minimally increasing the inclusion criteria to: a) more than one visit with ICD codes 354.0 or 354.1, or b) subsequent carpal tunnel surgery, would exclude 77 percent (34/44) of the "false" OCTS claims. However, 32 percent (64/201) of the true claims (with physician diagnosis) would also be excluded.

Diagnostic Coding Error Study—In order to determine how much misclassification by diagnosis exists in the data base, an extract was made of all medical bills with a CPT procedure code of 64721 (carpal tunnel decompression surgery) for the time period 1984–87. There were 2,507 unique carpal tunnel surgery claims with 212 associated ICD codes. A classification system (Table 5) was developed to assign the various diagnoses as: 1) CTS with ICD codes 354.0 or 354.1; 2) CTS of traumatic origin; or 3) all other ICD codes. This misclassification study is based on the assumption that all surgeries were indeed cases of CTS. If data were extracted from the system based entirely upon ICD codes 354.0 or 354.1, only 71.6 percent (1,793/2,507) of CTS claims would be captured. Only 3.4 percent (86/2,507) of claims that had received carpal tunnel surgery had diagnosis codes suggesting CTS of traumatic origin. Other diagnosis and data entry errors comprised 25 percent (628/2,421) of CTS surgery claims.

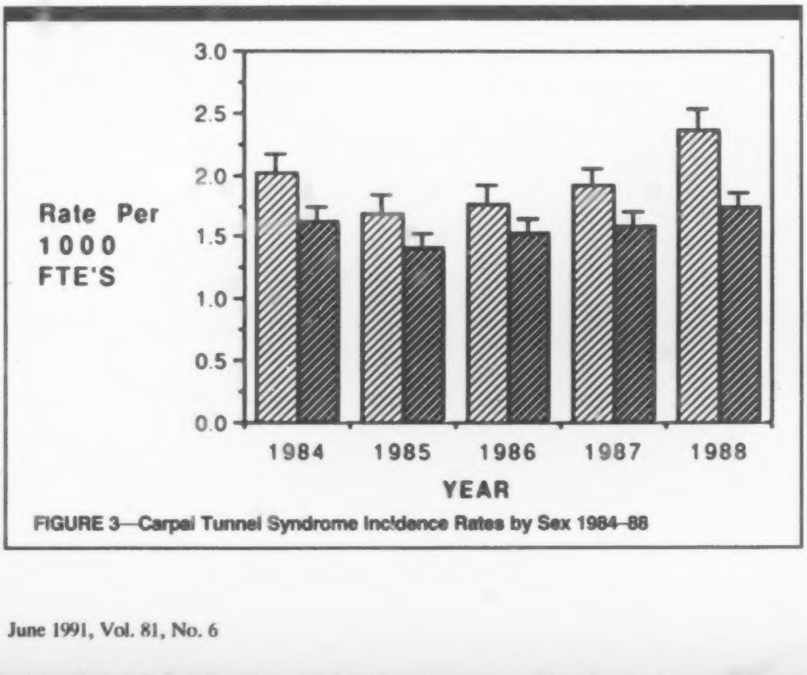

FIGURE 3—Carpal Tunnel Syndrome Incidence Rates by Sex 1984–88

TABLE 2—Rates and Cases of Carpal Tunnel Syndrome by Washington Industrial Classification (WIC), 1984–88 (sorted by rate)

Industrial Class Description	Cases N	Rate/FTE 1000	Rate Ratio	95% Confidence Interval
Oyster, crab, clam packing	50	25.7	14.8	(11.2, 19.5)
Meat, poultry dealers	132	23.9	13.8	(11.6, 16.4)
Packing house	55	18.5	10.6	(8.1, 13.8)
Fish canneries processing	124	18.2	10.5	(8.8, 12.5)
Carpentry	37	11.3	6.5	(4.7, 9.0)
Fruit & vegetable canning	55	10.2	5.8	(4.5, 7.6)
Egg production	30	9.8	5.6	(3.9, 8.1)
Box, shook, pallet bin manufacturing	25	9.3	5.4	(3.6, 8.0)
Sawmills, operation & maintenance	139	9.3	5.3	(4.5, 6.3)
Foundries, steel casting	37	9.0	5.2	(3.8, 7.2)
Logging operations	143	8.9	5.1	(4.4, 6.1)
Wallboard installation	43	8.4	4.8	(3.6, 6.5)
Roofwork, all types	48	8.0	4.6	(3.4, 6.1)
Boat building repair	36	7.7	4.4	(3.2, 6.1)
Plywood manufacturing	54	6.8	3.9	(3.0, 5.1)
Plastic goods manufacturing	121	6.5	3.7	(3.1, 4.5)
Food sundries manufacturing/processing	30	6.4	3.7	(2.6, 5.3)
Wood products manufacturing & assembly	53	6.4	3.7	(2.8, 4.8)
Lumber remanufacturing	26	6.4	3.7	(2.5, 5.4)
Building construction iron/steel/concrete	102	6.3	3.5	(2.9, 4.2)

TABLE 3—Rates and Cases of Carpal Tunnel Syndrome by Washington Industrial Classification (WIC), 1984–88 (sorted by number of cases)

Industrial Class Description	Cases N	Rate/FTE 1000	Rate Ratio	95% Confidence Interval
Convalescent & nursing homes	241	3.1	1.8	(1.6, 2.0)
Grocery/meat stores combination retail	215	3.7	2.1	(1.9, 2.4)
Class H restaurants	207	1.5	0.8	(0.7, 1.0)
Restaurants	170	2.0	1.2	(1.0, 1.4)
Fruit packing	158	5.5	3.1	(2.7, 3.7)
Logging operations	143	8.9	5.1	(4.4, 6.1)
Sawmills, operation & maintanence	139	9.3	5.3	(4.5, 6.3)
Meat, poultry dealers	132	23.9	13.8	(11.6, 16.4)
Fish canneries processing	124	18.2	10.5	(8.8, 12.5)
Plastic goods manufacturing	121	6.5	3.7	(3.1, 4.5)
Building construction iron/steel/concrete	102	6.0	3.5	(2.9, 4.2)
Clerical NOC	92	0.2	0.1	(0.1, 0.1)
Printing, lithography	80	2.9	1.7	(1.3, 2.1)
Fast food, drive-ins	74	1.0	0.6	(0.4, 0.7)
Orchards, fruit	72	1.1	0.6	(0.5, 0.8)
State employees	71	1.8	1.0	(0.8, 1.3)
Clothing manufacturing	71	5.0	2.9	(2.3, 3.7)
Higher education institutions	66	0.4	0.2	(0.2, 0.3)
Electric wiring in buildings	65	1.5	0.8	(0.7, 1.1)
Auto, truck, sales agencies	65	1.4	0.8	(0.6, 1.0)

TABLE 4—Validation Study Using Physician Diagnosis of OCTS

	Physician Diagnosis		
Inclusion Criteria	(Yes)	(No)	
One visit and no surgery	64	34	98
>One visit or subsequent surgery	137	10	147
Total	201	44	245

Discussion

Although findings from a number of recent case-control studies demonstrate a strong association between workplace exposures (high repetition, high force, vibration) and carpal tunnel syndrome,[7–11] this is the first population-based study to report age-, sex- and industry-specific incidence rates of OCTS. The mean age and female/male gender ratio (37.4 years; F:M = 1.2:1) of the OCTS population reported here differ dramatically from that reported for non-occupational carpal tunnel populations (51 years; F:M = 3:1).[12,13] These clear demographic differences between the occupational and non-occupational CTS populations, as well as the magnitude of numbers reported here, would appear to support the concept that workplace exposures may be responsible for a substantial proportion (up to 47 percent)[1] of physician-reported carpal tunnel cases.

The highest incidence rate reported here for shellfish packing (25.7 claims/1,000 FTEs) is similar to the highest reported incidence rates of "potential" carpal tunnel cases using Wisconsin WC data.[3] Industry-specific rates would be expected to vary according to the distributions of industries in each state or region. Some of the highest rates reported here are very characteristic of Washington State (seafood processing and canning, logging and wood products manufacturing, boat building), whereas others are represented in many regions and could be used for comparison (meat and poultry processing, carpentry, fruit and vegetable canning, egg production, foundries, wallboard installation, roofing, plastics manufacturing, and construction).

Industries reported in Table 3, sorted by total numbers of cases, represent more modest incidence rates and rate ratios. Rates for some of these industries (retail groceries, clothing manufacturing, printing and lithography, and electric wiring) may involve relatively weak CTS hazards, or may represent dilution of high exposure jobs by increased job diversification in some industries. Others with very high numbers of claims but non-significant rate ratios (e.g. restaurants) or difficult to explain exposures (e.g. convalescent and nursing homes) require further investigation.

Although video display terminal (VDT) operation has been associated with cumulative trauma disorders,[14] its relationship to OCTS has not been documented clearly in the literature. The industrial classes reported here whose claims may be related to VDT exposure (e.g. clerical, state employees, higher education employees) have non-elevated rate ratios. As these industrial classes represent a wide range of occupations, an association between VDT use and CTS may be diluted. Underreporting of cases in pre-

TABLE 5—Diagnostic Coding Errors for OCTS

71.6%	CTS diagnosis codes 354.0 and 354.1
3.4%	Probable CTS secondary to trauma (fracture, burn)
25.0%	ICD codes not specific for OCTS Miscellaneous data entry errors for OCTS

dominantly female job titles might be an alternative explanation for these lower than expected rates.

Rates among females increased significantly during the study period, while rates among males remained stable. The trends could be due to prior underreporting of claims among females or to a shift of women into higher exposure jobs. Because the gender-specific rates reported here depend on extrapolated rather than direct denominators, these conclusions should be validated in other studies.

Potential pitfalls in the use of workers' compensation[15] and other administrative data bases[16] to generate epidemiologic research have been well documented. Serious underreporting of OCTS cases to workers' compensation bureaus,[1,15] a lack of specificity in the current US Department of Labor coding system,[3] and difficulty generating direct denominators[17] present formidable obstacles that will require public policy changes before improvement is realized. We were able to mitigate the effects of the latter two problems by use of physician-generated ICD billing codes and worker hours reported by WIC, respectively.

Misclassification and coding errors are other serious problems in using workers' compensation data. Incidence rates for OCTS presented were subject to error by both overcounting and undercounting. We were able to validate that only 82 percent of incident claims are true cases of carpal tunnel syndrome by documented physician diagnosis in the medical records. This potential source of overcounting, however, was non-differential by age, sex, or year, and using more or less restrictive inclusion criteria for incident claim definition did not reduce the degree of misclassification.

An equally serious source of potential undercounting of incident claims was also encountered, particularly in regard to ICD coding errors. Only 72 percent of unique claims that were associated with carpal tunnel surgery had an ICD code (354.0 or 354.1) compatible with carpal tunnel syndrome. Another 3.4 percent of claims had diagnosis codes that were related to CTS of traumatic origin, but 25 percent of claims had either non-specific diagnoses or erroneous diagnostic codes. The latter cases would not have been captured in this study. No gains in precision could be derived from applying these estimates of misclassification to the OCTS incidence rates.

The WIC system is specific to Washington State and is not directly comparable to the national Standard Industrial Classification (SIC) coding system. We were unable to generate reliable SIC code-specific OCTS rates because employee hours are not reported to L&I by SIC code. The WIC system differs from the SIC system in that the former sets aside a separate classification for some occupations such as clerical workers regardless of the employers principal industry type. However, the WIC and SIC systems of classification are sufficiently similar to allow other investigators indirect comparability.

On balance, and with more complete understanding of the limitations of workers' compensation[15] and administrative data bases,[16] workers' compensation data can be useful in generating population-based data for the purposes of surveillance, forecasting trends, and resource targeting for loss control and prevention. The use of other surveillance systems, dependent on direct physician or laboratory reporting,[18,19] could provide complementary information. Workers' compensation data might be particularly useful in the investigation of occupational conditions for which no other good surveillance systems exist, such as OCTS, morbid head injuries, labor injuries among minors, sexual assaults,[20] or for conditions related to mandatory reporting, such as lead poisoning.[21] □

Acknowledgments

This work was funded by the Accident and Medical Aid Funds of the State of Washington, Department of Labor and Industries. These research monies are targeted to reducing the incidence and disability related to occupational injuries and illnesses. Special thanks are due to Barbara Silverstein, PhD, for critical review of the manuscript, and to Melinda Fujiwara for preparation of the manuscript.

This work was presented in part at the American Academy of Neurology, Miami, Florida, May 1990.

References

1. Cummings K, Maizlish N, Rudolph L, Dervin K, Ervin A: Occupational disease surveillance: Carpal tunnel syndrome. MMWR 1989; 38:485–489.
2. Masear VR, Hayes JM, Hyde AG: An industrial cause of carpal tunnel syndrome. J Hand Surg 1986; 11A:222–227.
3. Hanrahan LP, Moll MB: Injury surveillance. Ch. VIII. *In*: Baker EL (ed): Surveillance in Occupational Health and Safety. Am J Public Health 1989; 79(Suppl):38–45.
4. US Dept of Health and Human Services: ICD–9–CM. International Classification of Diseases, 9th Rev Clinical Modification. DHHS Pub. No. (PHS) 80–126. Washington, DC: DHHS, 1980.
5. Breslow NE, Day NE: Statistical Methods in Cancer Research. Vol. II: The Design and Analysis of Cohort Studies. IARC Sci. Pub. No. 82; 91–103. Lyons, France: IARC, 1987.
6. Rothman KJ: Modern Epidemiology. Boston/Toronto: Little, Brown, 1986; 153–175.
7. Armstrong TJ, Chaffin DB: Carpal tunnel syndrome and selected personal attributes. JOM 1979; 21:481–486.
8. Cannon LJ, Bernacki EJ, Walter SD: Personal and occupational factors associated with carpal tunnel syndrome. JOM 1981; 23:255–258.
9. Franklin GM, Boteler D, Nelson L: Occupational carpal tunnel syndrome in an Oregon neurology practice. Am J Epidemiol 1984; 120:493.
10. Silverstein BA, Fine LJ, Armstrong TJ: Occupational factors and carpal tunnel syndrome. Am J Ind Med 1987; 11:343–358.
11. Wieslander G, Norback D, Gothe CJ, Juhlin L: Carpal tunnel syndrome and exposure to vibration, repetitive wrist movements, and heavy manual work: A case-referent study. Br J Ind Med 1989; 46:43–47.
12. Stevens JC, Sun S, Beard CM, O'Fallon WM, Kurland LT: Carpal tunnel syndrome in Rochester, Minnesota, 1961 to 1980. Neurology 1988; 38:134–138.
13. Stewart JD, Aguayo AJ: Compression and entrapment neuropathies. *In*: Dyck PJ, Thomas PK, Lambert EH, Bunge R (eds): Peripheral Neuropathy (Vol. 2): Philadelphia: WB Saunders Co, 1984; 1435–1437.
14. Heyer N, Checkoway H, Daniell W, Horstman S, Camp J: Self-reported musculoskeletal symptoms among office video display terminal operators. *In*: Sakurai H, Okazaki I, Omae K (eds): Occupational Epidemiology. Excerpta Medicine International Congress Series 889: Excerpta Medica, 1990; 255–258.
15. Schwartz E: Use of workers' compensation claims for surveillance of work-related illness—New Hampshire, January 1986–March 1987. MMWR 1987; 36:713–720.
16. Connell FA, Diehr P, Hart LG: The use of large databases in health care studies. Annu Rev Public Health 1987; 8:51–74.
17. Tanaka S, Seligman P, Halperin W, Thun M, Timbrook CL, Wasil JJ: Use of workers' compensation claims data for surveillance of cumulative trauma disorders. JOM 1988; 30:488–492.

18. Rutstein DD, Mullan RJ, Frazier TM, Halperin WF, Melius JM, Sestito JP: Sentinel health events (occupational): A basis for physician recognition and public health surveillance. Am J Public Health 1983; 73:1054–1062.
19. Matte TD, Baber EL, Honchar PA: The selection and definition of targeted work-related conditions for surveillance under SENSOR. Ch. V. *In*: Baker ET (ed): Surveillance in Occupational Health and Safety. Am J Public Health 1989; 79(Suppl):21–25.
20. Seligman PJ, Newman SC, Timbrook CL, Halperin WE: Sexual assault of women at work. Am J Ind Med 1987; 12:445–450.
21. Seligman PJ, Halperin WE, Mullan RJ, Frazier TM: Occupational lead poisoning in Ohio: Surveillance using workers' compensation data. Am J Public Health 1986; 76:1299–1302.

Developmental, Learning, and Emotional Problems Affect 10.7 Million American Children

One in five American children has had a developmental delay, learning disability, or emotional problem according to a survey by the National Center for Health Statistics. The 1988 National Health Interview Survey of Child Health found such disorders among the most prevalent chronic conditions of childhood and adolescence, with a total of 10.7 million children affected. By the teen years, one in four children had experienced one of these disorders and, among male teenagers, almost three in 10 have been affected.

A report on the survey *Developmental, Learning and Emotional Problems: Health of Our Nation's Children, United States, 1988,* states that 4 percent of all children 17 years of age and under (2.5 million) had delays in growth or development; 6.5 percent among children aged 3–17, 3.4 million (6.5 percent) had learning disabilities and 7 million (13.4 percent) have had an emotional or behavioral problem that lasted three months or more or required psychological treatment.

About half of the children with a developmental delay had received treatment or counseling. More than three fourths of those with learning disabilities or emotional problems had received treatment or counseling, according to the parents reporting in the survey. Parents who reported that their child had a learning, developmental or emotional disorder were asked whether the condition made it necessary for the child to attend special classes, or a special school, or get special help at school. About a quarter of children with delays in growth or development and those reported to have emotional or behavior problems received special educational help. About 70 percent of those with learning disabilities received educational assistance for their disabilities.

In relation to family structure, young people from single-parent families or stepfamilies were two to three times more likely to have had emotional or behavior problems than those from two-parent homes. The prevalence of childhood emotional and behavioral problems showed significant variation across family income groups and minority populations with children from less advantaged backgrounds standing a somewhat greater chance of exhibiting such problems. Black and Hispanic parents were less likely than White non-Hispanic parents to report their children's developmental delays or emotional problems, suggesting that emotional and behavioral problems may be underreported by minority parents, when the data from this nationwide household interview survey are compared to data from other sources. Teacher reports and school records, for example, suggest that minority children are over-represented among special education and remedial instruction classes.

The 1988 National Health Interview Survey of Child Health was a survey of 17,100 children 17 years of age and under, residing in the home, and was based on interviews with an informed adult member of the family, usually the mother.

To obtain a copy of the child health report, *Developmental, Learning and Emotional Problems,* contact NCHS, CDC/PHS/DHHS, 6525 Belcrest Road, Hyattsville, MD 20782; (301) 436-8500.

Socioeconomic Status and Survival from Soft-Tissue Sarcomas: A Population-Based Study in Northern Italy

Giovannino Ciccone, MD, Corrado Magnani, MD, Luisa Delsedime, MD, and Paolo Vineis, MD

ABSTRACT

Background and *Purpose:* Differential prognosis among cancer patients according to socioeconomic status (SES) has been reported. We analyzed survival from soft tissue sarcomas (STS) according to different SES indicators.

Methods: We followed up all the adult patients with a new diagnosis of STS occurring between 1.1.1981 and 31.12.1983 in an area of Northern Italy (N = 86).

Results: The overall three-year survival rate was 57 percent. After adjustment for confounders, both low education and blue collar jobs were negatively associated with survival.

Conclusions: The results suggest that patients of low SES have a poorer prognosis for STS. (*Am J Public Health.* 1991;81:747–749)

Introduction

The five-year relative survival rates from soft-tissue sarcomas (STS) were about 60 percent in the United States in 1979–84.[1] Several studies have described the clinical and pathological determinants of survival from STS.[2–5]

In recent years, considerable attention has been paid to differential prognosis among cancer patients according to socioeconomic status (SES), particularly for breast and colon cancer.[6–9]

Differences in survival are interpreted as related to earlier access to diagnosis and therapy of higher social class patients.

We are not aware of previous investigations of the role of socioeconomic factors as predictors of survival among STS patients. The present investigation is based on the follow-up of all patients with a new diagnosis of STS among residents of a geographic area of Northwestern Italy.

Methods

A population-based case-control study of STS in the provinces of Novara, Vercelli, and Alessandria in Northwestern Italy was carried out between January 1, 1981 and December 31, 1983 among residents of 20 or more years of age to investigate the role of exposure to phenoxy herbicides in the etiology of STS.[10] Eighty-six subjects were enrolled, after histologic confirmation of the diagnosis by two independent pathologists.[10] The vital status and cause of death of all 86 subjects were ascertained during 1987 through correspondence with the municipalities where they resided at the time of diagnosis. The median follow-up duration was 38 months (mean 34.5; range 0–70).

In 1988 a subset of slides (n = 58) was reviewed by a different group of pathologists to evaluate each case according to a histopathological grading classification proposed by French authors.[3,4]

Within the case-control study, 18 of the 86 original cases could not be interviewed. Details on the questionnaire and the procedures involved are given elsewhere.[10] The following variables were analyzed as potential predictors of survival: age, sex, marital status, education, smoking habits, occupation, exposure to phenoxy herbicides, type of hospital of diagnosis (large public hospital vs small or private hospital), and site of origin, histotype, and histopathological grading of the sarcoma.

The analyses were performed using SAS programs.[11,12] Estimates of survival were obtained using the Kaplan-Meier product-limit method[13]; the statistical significance of the difference between survival curves was evaluated through the log rank test.[14] A multivariate analysis was performed including different SES indicators and other covariates; the Cox proportional hazards model was used.[15] Proportionality of risk over time was assessed for each variable introduced in the models through the plots of the log (−log) of the survival distribution function on the log of time.

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TABLE 1—Proportion of Deceased Patients at the end of Follow-up according to Selected Variables Collected from Municipality Rosters or Hospital Records (whole series of 86 cases) and at Interview (68 cases)

Variables	Deceased n (%)	Total n
Success of interview		
yes	30(44)	68
no	11(61)	18
Age (years)		
<45	8(36)	22
45–54	7(41)	17
55–64	8(50)	16
65+	18(58)	31
Sex		
males	15(37)	41
females	26(58)	45
Hospital of diagnosis		
large, public	25(50)	50
small or private	16(44)	36
Years of school		
<=5	25(51)	49
6+	4(24)	17
missing	1(50)	2
Marital status		
Married	21(47)	45
Unmarried	4(40)	10
Other	6(46)	13
Smoking		
yes	15(47)	32
no	16(46)	35
missing	0 (0)	1
Main Occupation		
Manual worker	14(52)	27
Farmer	14(58)	24
Others	3(18)	17
Exposure to herbicides		
yes	7(58)	12
no	24(43)	56

TABLE 2—Multivariate Analysis (Cox's proportional hazards model): Role of Education and Occupation in Influencing Survival among the 68 Interviewed Cases*

	Model and SES indicators	Coefficient	SE
I	years of school <=5	1.33	0.61
II	manual workers	0.90	0.39
	farmers	0.37	0.43
III	years of school <=5	1.06	0.64
	manual workers	0.65	0.42
	farmers	0.15	0.45

*Each model includes also sex, age, and histopathological grading.

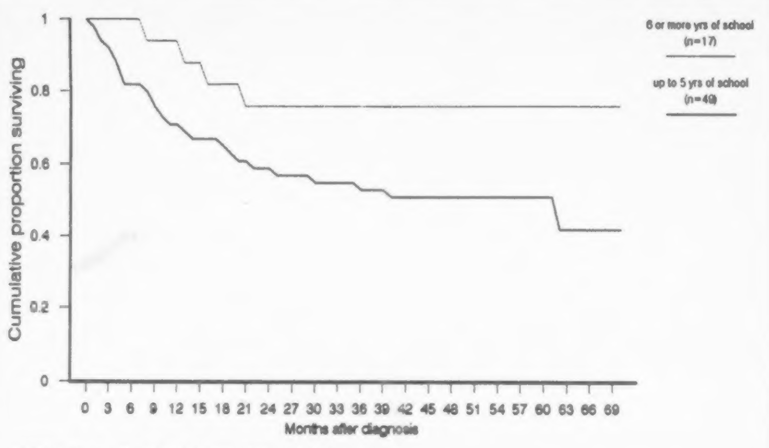

FIGURE 1—Survival from STS by Education

Results

The overall proportion of patients alive three years after a diagnosis of sarcoma of soft tissues was 57 percent.

The Appendix shows the distribution of the patients by site of the tumor, histology, and grading, according to vital status at the end of follow-up. Only two-thirds of cases could be reviewed for histopathologic grading: there is evidence that such classification is likely to be an important prognostic factor for STS.

Table 1 reports the distribution of several characteristics of the patients by vital status at the end of follow-up.

By univariate analysis, the less educated patients and unskilled workers (manual workers or farmers vs others) had poorer prognoses. Figure 1 shows the difference in the proportion surviving according to education. A similar pattern appears comparing survival experiences according to the main occupation.

To take into account the strong association between low education and unskilled jobs and the role played by other covariates in influencing survival, a multivariate analysis was performed, fitting three models. In all models estimates of different SES indicators were adjusted for sex, age, and histopathologic grading. Table 2 shows that low education is negatively related to survival; among blue collar workers, a poorer prognosis is evident for manual workers in industry but not for farmers.

Discussion

Differences in cancer survival according to socioeconomic status of the patient have already been observed for other cancers, both when comparing different categories of workers (i.e. blue vs white collar)[8] and when comparing different ethnic groups.[9] The differences seem to affect particularly breast and colorectal cancers which, at least in theory, could be more influenced by early diagnosis. In a study based on the Swedish Cancer Registry,[8] the following tentative interpretations for the observed social class differences were proposed: earlier detection in higher social class, with improvement of prognosis; earlier detection in higher social class, without improvement of prognosis (lead time bias); host factors, associated with social class, which influence the response to treatment; biological properties of the tumors arising in different social classes.

There is no evidence in favor of host factors or biologic differences in the tumors. Early detection seems to be more reasonable, but it is difficult to disentangle pure "lead time" (i.e. earlier diagnosis without real improvement of survival) and effectiveness of treatment.

Apart from histopathologic grading, only socioeconomic indicators showed an association with survival rates, with patients of low socioeconomic groups showing a worse prognosis. This observation does not rule out a more complex interpretation involving other factors not considered in the present analysis. Although our findings are based on a relatively small number of cases, they cannot be easily explained by bias, since the follow-up was successful for 100 percent of the 86 patients; SES was determined on *a priori* categorization of education and occupation; follow-up procedures were completely blind as to the predictors used in the analyses; differences in survival between patients of different socioeconomic groups were not confounded by age, sex, or tumor grading.

Unfortunately, we had incomplete information about clinical variables, such as stage of cancer at first symptoms or signs or type of treatment. Separate analysis according to histologic type is of little help because there are too few cases in each group. Although they show different probabilities of survival, ranging from zero through almost 100 percent, there is no obvious grouping of histologic types which could make a comparison between social classes easier. The histopathologic grading, a good prognostic factor, was available for only two-thirds of our cases.

The present report, like those cited earlier, suggests the existence of inequalities in health, associated with patient socioeconomic status. More analytical studies to control from a pure "lead time" effect are in order. □

APPENDIX—Proportion of Decreased Patients at the End of Follow-up by Site, Histotype,* and Grading of the Soft-tissue Sarcoma.

	Deceased n	Total n
Site of the tumor		
Head and neck	3	8
Retroperitoneal	5	8
Upper limb	4	13
Lower limb	12	23
Thorax	8	13
Abdomen	0	2
Pelvis	4	6
Trunk, unsp.	1	1
Other and unsp.	4	12
Histotype		
Fibrohistiocytic tumors	7	14
Dermatofibrosarcoma protuberans	0	3
Malignant histiocytoma	7	11
Tumors of adipose tissue	2	10
Liposarcoma	0	3
Myxoid liposarcoma	2	7
Tumors of muscle tissue	6	9
Leiomyosarcoma	4	7
Rhabdomyosarcoma	2	2
Tumors of blood vessels	4	17
Angiosarcoma	1	1
Kaposi sarcoma	1	13
Malignant hemangiopericytoma	2	2
Other vascular malignant tumors	0	1
Tumors of synovial tissue	2	3
Synovial sarcoma	2	3
Tumors of peripheral nerves	5	7
Malignant Schwannoma	4	4
Other mal. tumors of peripheral nerves	1	3
Tumors of paraganglionic structures	1	1
Malignant tumors of nonchromaffin tissue	1	1
Tumors of cartilage and bone forming tissue	2	4
Chondrosarcoma of soft parts	2	4
Tumors of uncertain histogenesis	1	2
Malignant cell tumor of soft parts	0	1
Chordoma	1	1
Unclassified	11	19
Histopathologic grading		
Grade I	6	26
Grade II	11	23
Grade III	7	9
Missing	17	28

*For the classification used, see reference 16.

Acknowledgments

This research was supported by Regione Piemonte (Ricerca Biomedica Finalizzata), by the Lega Italiana per la Lotta contro i Tumori (sezione provinciale di Alessandria), and by CSI-Piemonte. In addition G. Ciccone received a fellowship from the Associazione Italiana per la Ricerca sul Cancro.

We acknowledge the heads of the pathology departments of the hospitals of Alessandria, Vercelli, Novara, Borgomanero, Casale M.to, Biella, and of the National Cancer Institute in Milan for access to their files. We thank Drs. A. Donna and R. Pisa for first review of the cases, and Prof. F. Mollo and his colleagues for the grading classification of the cases. We also thank Lucia Nonnato for technical assistance.

References

1. National Cancer Institute: 1987 Annual Cancer Statistics Review. Washington, DC: US Department of Health and Human Services, NCI, Division of Cancer Prevention and Treatment, 1988.
2. Rooser B, Attewell R, Berg NO, Rydholm A: Prognostication in soft tissue sarcoma—A model with four risk factors. Cancer 1988; 61:817–823.
3. Trojani M, Contesso G, Coindre JM, Rouesse J, Bui NB, de Mascarel A, Goussot JF, David M, Bonichon F, Lagarde C: Soft-tissue sarcomas of adults: Study of pathological prognostic variables and definition of a histopathological grading system. Int J Cancer 1984; 33:37–42.
4. Ueda T, Aozasa K, Tsujimoto M, Hamada H, Hayashi H, Ono K, Matsumoto K: Multivariate analysis for clinical prognostic factors in 163 patients with soft tissue sarcoma. Cancer 1988; 62:1444–1450.
5. Mandard AM, Petiot JF, Marnay J, *et al:* Prognostic factors in soft tissue sarcomas. A multivariate analysis of 109 cases. Cancer 1989; 63:1437–1451.
6. Berg J, Ross R, Latourette H: Economic status and survival of cancer patients. Cancer 1977; 39:467–477.
7. Keirn W, Metter G: Survival of cancer patients by economic status in a free care setting. Cancer 1985; 55:1552–1555.
8. Vagero D, Persson G: Cancer survival and social class in Sweden. J Epidemiol Community Health 1987; 41:204–209.
9. Young JL Jr, Gloeckler Ries L, Pollack ES: Cancer patient survival among ethnic groups in the United States. JNCI 1984; 73:341–352.
10. Vineis P, Terracini B, Ciccone G, Cignetti A, Colombo E, Donna A, Maffi L, Pisa R, Ricci P, Zanini E, Comba P: Phenoxy herbicides and soft-tissue sarcomas in female rice weeders. Scand J Work Environ Health 1987; 13:9–17.
11. SAS Institute Inc: SAS User's Guide: Statistics, Version 5 Edition. Cary, NC: SAS Institute Inc, 1985.
12. Harrell FE Jr: The PHGLM procedure. *In:* SAS Institute Inc. SUGI Supplemental Library User's Guide, Version 5 Edition. Cary, NC: SAS Institute Inc, 1986; 437–466.
13. Kaplan EL, Meier P: Nonparametric estimation from incomplete observations. J Am Stat Assoc 1958; 53:457–481.
14. Mantel N: Evaluation of survival data and two new rank order statistics arising in its consideration. Cancer Chemother Rep 1966; 50:163–170.
15. Cox DR: Regression models and life-tables. J R Stat Soc 1972; Series B 34:187–220.
16. Enzinger FM, Weiss SW: Soft Tissue Tumors. 2nd Ed. St. Louis: C. V. Mosby Company, 1988.

Public Health Briefs

Pesticide Poisoning Surveillance through Regional Poison Control Centers

Debra Kay Olson, RN, MPH, Lynn Sax, Paul Gunderson, PhD, and Leo Sioris, PharmD

ABSTRACT

The purpose of this study is to describe pesticide exposure in the population of callers to Minnesota Regional Poison Centers. Case files from 1988 reporting pesticide exposure to humans were identified in cooperation with the Minnesota Center for Health Statistics. Data analysis was conducted by computer using SAS statistical package.

Of the 1,428 case files indicating pesticide as the primary substance of exposure to Minnesota residents, a mean age of 5 years (range, one month to 85 years) was identified; 50 percent of all cases were below age 3 years. Males accounted for 1.3 times as many cases as females.

Insecticide was identified in the largest percentage of case files (74 percent) followed by herbicide (12 percent), rodenticide (11 percent) and fungicide-nonmedicinal (3 percent). Ingestion was the most common route of exposure; 85 percent of all calls originated from a residence.

While insecticides are still the most common types of pesticide call, herbicide has surpassed insecticide in production and sales in the US. In this study, herbicide type exposure calls present a much different picture than other pesticide types.

The usefulness of poison control centers for examination of pesticide poisoning is explored. Since reporting occurs coincidental with the exposure and its associated symptoms, each pesticide poisoning report could potentially serve as a true sentinel health event. (*Am J Public Health.* 1991;81:750–753)

Introduction

Pesticides represent a large group of chemicals which in general usage are described in terms of the organism they are intended to kill.[1–3] As the use of these chemicals has unfolded, some disquieting phenomena have been observed. A 1984–85 Nebraska study found an annual incidence rate of pesticide-related illness of 1.35 cases per 10,000 population.[4] Lymphoma and leukemia have been associated with higher death rates among Midwestern farmers in regions of high pesticide use[5,6] and Reeves[7] reported 15 children ages 2 to 17 years as having blood dyscrasias developed shortly after inhalation exposure to household insecticides. Pesticides account for a small but important number of acute human poisonings, particularly in a state with substantial use of pesticides in both rural and urban areas, such as Minnesota.[8,9]

Methods

The purpose of this study was to describe pesticide exposure in the 1988 population of callers to Minnesota Regional Poison Control Centers located at Hennepin County Medical Center and St. Paul-Ramsey Medical Center. Centers document exposure data utilizing the Cooperative Poison Center Report Form as designed by the American Association of Poison Control Centers (AAPCC). Data from these report forms were processed through each Center's computer system and sent to the AAPCC's national data collection project in Denver, Colorado. Data tapes containing all of the 1988 data for each Regional Center were then returned to the Minnesota Center for Health Statistics (MCHS) for collation, formatting, and analysis.

In cooperation with the MCHS all Minnesota resident case files from 1988 reporting pesticide exposure to humans were identified. All calls regarding human contact with a chemical coded by the AAPCC to be a herbicide, insecticide, rodenticide, or fungicide (nonmedicinal) were selected. A total of 2,209 calls were classified by the AAPCC system as pesticide exposure calls for Minnesota in 1988, 4.3 percent of all human exposure calls to Minnesota. Of these calls, 1,428 identified pesticides as the primary substance and 31 call files identified pesticides as a secondary substance of human exposure to Minnesota residents. The remaining 750 calls were incomplete data files or lacked the designation as a Minnesota resident, thus were not available for secondary analysis.

The following descriptive analysis applies to the 1,428 call files indicating pesticide as the primary substance of exposure to Minnesota residents in 1988. These data were derived from self-reported information. The use of poison control caller files imposes the following limitations upon the data: an unknown number of pesticide exposure events may occur for which no call was made; voluntary reporting of data via telephone is verified only by

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TABLE 1—Insecticide Exposure Calls by Primary Substance Category, Minnesota, 1988

Substance	Frequency	Percent
Organophosphate only	220	20.8
Organophosphate in combination with carbamate	71	6.7
Organophosphate in combination with chlorinated hydrocarbon	15	1.4
Organophosphate in combination with other pesticide	7	0.7
Chlorinated hydrocarbon only	116	10.9
Chlorinated hydrocarbon in combination with other pesticide	6	0.6
Borates/boric acid (pesticide only)	29	2.7
Carbamate only	189	17.8
Carbamate in combination with other pesticide	6	0.6
Veterinary pesticides (for pets - flea collars, etc.)	49	4.6
Piperonyl butoxide and pyrethrins in combination (without organophosphate or carbamate)	80	7.5
Pyrethrins only (alone)	59	5.6
Rotenone	5	0.5
Piperonyl butoxide only (alone)	2	0.2
Insect repellants (excluding capsicum containing products)	163	15.4
Other	29	2.7
Unknown	14	1.3
Total	1060	100.0

SOURCE: Hennepin Regional Poison Center and Minnesota Regional Poison Center. Secondary analysis performed by the Minnesota Center for Health Statistics and Public Health Nursing, University of Minnesota.

follow-up phone calls by poison center specialists; data acquired by telephone are limited to those who have access to telephones and knowledge of the poison center service; and by AAPCC definition exposure relates only to suspected contact with any substance(s) which, when inhaled, ingested, applied to, injected into, or developed within the body, may cause damage to structure or disturbance of function to living tissue. An actual poisoning event can only be verified through review of medical outcome.

Results

Insecticide was identified in the largest percentage of case files for Minnesota (1,060 cases, 74 percent) followed by herbicide (168 cases, 12 percent), rodenticide (155 cases, 11 percent), and fungicide-nonmedicinal (45 cases, 3 percent). Of the insecticide cases, those containing organophosphates (alone or in combination) were reported most often (Table 1).

Carbamates were reported in 10 cases of fungicide exposure. Of the remaining fungicide cases, 19 were associated with phthalimide, three with non-mercurial fungicides, and 13 unknown/other.

Herbicide cases were most frequently associated with 2,4-D (2,4-dichlorophenoxyacetic acid) or 2,4,5-T (2,4,5-trichlorophenoxyacetic acid) (78 cases, 46 percent) and triazine herbicides (10 cases, 6 percent). Diquat was associated with 13 cases, carbamate with four cases, and other or unknown with 63 cases.

Rodenticide exposure calls were primarily associated with anticoagulant types (128 cases, 83 percent). Strychnine was associated with three rodenticide exposure calls and the remaining 24 rodenticide cases were other/unknown.

Of the 1,428 cases, a mean age of 5 years (range, one month to 85 years) was identified; 50 percent of all calls were regarding children under the age of 3 years (Table 2). The 13–17 years category accounted for the least number of cases overall. The 18+ years old category accounted for 53 percent of all herbicide-related calls.

Male to female ratio was 1.3:1 ± 0.2 for all pesticide-related calls, as well as for individual pesticide types, except herbicides, where males represented twice the number of cases as females.

Ninety-nine percent of all pesticide calls were reported as unintentional poisonings. General calls (not otherwise specified) accounted for 94 percent (1,323 calls) of the unintentional calls while occupational-related exposure made up 4 percent (62 calls).

Calls originating from a residence accounted for 85 percent of all pesticide poisoning calls. Health care facilities accounted for 11 percent and work place for 3 percent.

Ingestion was the most common route of exposure overall, and for each pesticide type (60 percent), except herbicide, where ingestion accounted for 39 percent and dermal exposure accounted for 45 percent of calls.

Pesticide exposure calls by two-hour interval present a bimodal distribution peaking between 10:00 am and 11:00 am (190 calls, 13 percent) and 6:00 pm and 7:00 pm (251 calls, 18 percent). Calls by two-hour interval by pesticide type show a similar bimodal distribution for each specific type of pesticide. The largest percent of pesticide-related calls occur in the months of May (16 percent), July (15 percent), and August (13 percent) (Table 3).

Thirty-three percent of the calls (468 calls) were documented as symptomatic. Two hundred eighty-eight of these were classified as symptomatic related to the pesticide exposure. By pesticide type, rodenticide calls reported the least number (3 percent) of calls as symptomatic related to the exposure.

Ninety-nine percent of all calls were associated with acute exposures. The majority of calls were managed at a non-health care facility (86 percent) while 12 percent were either managed at (107 calls) or referred to (69 calls) a health care facility. More fungicide cases (93 percent) were managed at a non-health care facility (e.g. managed at home) than any other pesticide type.

Discussion

Since 1986, pesticides have accounted for approximately 4.3 percent of all human exposure calls reported to Minnesota Regional Poison Centers. This is consistent with the percent of calls associated with pesticides nationally as reported by the AAPCC.[8] As such, the state data reported here represent but a small portion of a much larger national picture.

Over the past 20 years in the US, organochlorine insecticides have lost favor because of their long-term health effects and persistence in the environment and have been surpassed in use by organophosphates and carbamates.[10] Acute toxicity related to organophosphate pesticides is usually of rapid onset thus symptoms can be more readily associated with acute exposure to this pesticide type. It is thus not unreasonable that exposure to organophosphate insecticides make up the greatest percent of calls (22 percent) in this study. This is also consistent with a Nebraska study where 25 percent of the emergency room visits and hospitaliza-

TABLE 2—Type of Primary Pesticide Exposure Calls by Age, Minnesota, 1988

	Fungicides		Herbicides		Insecticides		Rodenticides	
Age (years)	Freq.	%	Freq.	%	Freq.	%	Freq.	%
<1	4	0.3	0	0.0	98	6.8	16	1.2
1	8	0.5	11	0.7	197	13.8	57	4.0
2	9	0.6	17	1.1	187	13.1	39	2.7
3	4	0.3	7	0.5	91	6.4	15	1.0
4	1	0.1	5	0.4	39	2.7	2	0.1
5	0	0.0	5	0.4	34	2.4	1	0.1
Unknown child								
2–5	0	0.0	5	0.4	19	1.3	1	0.1
6–12	2	0.1	15	1.0	62	4.3	2	0.1
13–17	1	0.1	8	0.5	17	1.2	2	0.1
18+	15	1.0	89	6.3	302	21.1	14	1.0
Unknown age	1	0.1	6	0.5	14	1.0	6	0.5
Total	45	3.1	168	11.8	1060	74.2	155	10.9

SOURCE: Hennepin Regional Poison Center and Minnesota Regional Poison Center. Secondary analysis performed by the Minnesota Center for Health Statistics and Public Health Nursing, University of Minnesota.

TABLE 3—Type of Primary Pesticide Exposure Calls by Month, Minnesota, 1988

Month	Fungicides	Herbicides	Insecticides	Rodenticides	Row	Total (%)
January	1	—	59	7	67	(4.7)
February	1	—	32	11	44	(3.1)
March	2	2	31	11	46	(3.2)
April	7	11	45	18	81	(5.7)
May	13	58	141	17	229	(16.0)
June	7	43	176	12	238	(16.7)
July	5	21	182	6	214	(15.0)
August	4	22	149	14	189	(13.2)
September	3	8	99	19	129	(9.0)
October	1	2	60	15	78	(5.5)
November	—	—	49	12	61	(4.3)
December	1	1	37	13	52	(3.6)
Total	45	116	1060	155	1428	(100.0)

SOURCE: Hennepin Regional Poison Center and Minnesota Regional Poison Center. Secondary analysis performed by the Minnesota Center for Health Statistics and Public Health Nursing, University of Minnesota.

tions for pesticide-related illness during the 1984–85 crop season were due to organophosphates.[4]

In our study herbicide type exposure calls present a much different picture than other pesticide types. They are typically associated with the adult (18+ years) male experiencing a dermal exposure. With the growing use of herbicides both in agricultural (175 percent increase from 25 years ago[2]) and urban settings the percentage of calls received (0.4 percent of all human exposure calls since 1985) in comparison to exposure potential appears low.[11–14]

Looking at the adult environments of work place (six calls) and type of exposure as occupational (18 calls), the difference in exposure for herbicides by age is not explained. It is still general unintentional residence exposures that account for the majority of herbicide calls in this study. Why this overrepresentation of adults for herbicide exposure calls occurs is an area for further research.

With 50 percent of all calls, examined in this study, reporting pesticide exposure to children under the age of three years, concern arises over long-term effects to this population group. Several authors have explored the long-term effects of such exposures on children.[2,15,16]

Chronic exposure to fungicides has been reported by Moses[2] as posing the greatest risk of cancer as compared to other pesticide types. Thus, while representing only 3 percent of the calls in this study, the cumulative effects of such exposures may have far greater implications.

Blanc and Olson[17] suggest that poison centers are ideally suited to occupational disease surveillance as used for early detection of disease, for timely identification of individual cases, and temporal trends. In this sense, each poison center report could potentially serve as a sentinel health event. Sentinel health events refer to a preventable disease, disability, or untimely death whose occurrence serves as a warning signal that the quality of preventive and/or therapeutic medical care may need to be improved.[18,19]

Acute pesticide poisoning reports, as used in this study, would meet the criteria used by Blanc, *et al*,[17,20] in their investigation of the usefulness of poison center data. Those exposures to pesticides with rapid onset of symptoms, such as in the case of organophosphates, are ideally suited for surveillance through such a system. Reporting occurs coincidental with the exposure and its associated symptoms, and there are no delays as in the case of retrospective surveys. In this sense, each pesticide poisoning report could potentially serve as a true sentinel health event for the state. ☐

Acknowledgments

The initial development of the statewide data system was funded by the Minnesota Regional Poison Center and the Medical Education Research Foundation at St. Paul-Ramsey Medical Center in cooperation with the Minnesota Department of Health, Minnesota Center for Health Statistics. Additional support was provided by the University of Minnesota, School of Public Health, Public Health Nursing and BRSG S 07 RR 055448, Biomedical Research Support Grant Program, Division of Research Resources, and National Institutes of Health grant. Analysis of pesticide poisoning exposure data was funded in part by the National Institute for Occupational Safety and Health, Occupational Health Nursing Research Training grant, Midwest Center for Occupational Health and Safety, and the Minnesota Department of Health.

References

1. Zenz C: Occupational Medicine: Principles and Practical Applications. Chicago: Year Book Medical Publishers, 1988.
2. Moses M: Pesticide-related health problems and farmworkers. AAOHNJ 1989; 37(3):115–130.
3. Ware GW: Pesticides: Theory and Application. San Francisco: W.H. Freeman, 1983.
4. Rettig BA, Klein DK, Sniezek JE: The incidence of hospitalizations and emergency room visits resulting from exposure to chemicals used in agriculture. Nebr Med J 1987; 7:215–219.
5. Weisenberger DD: Lymphoid malignancies in Nebraska: A hypothesis. Nebr Med J 1985; 70:300–305.
6. American Medical Association, Council on Scientific Affairs: Council Report: Cancer risk of pesticides in agricultural workers. JAMA 1988; 260:959–966.
7. Reeves JD: Household insecticide-associated blood dyscrasias in children (letter). Am J Pediatr Hematol/Oncol 1982; 4:438–439.
8. Litovitz TL, Schmitz BF, Matyunas N, Martin TG: 1987 Annual Report of the American Association of Poison Control Centers National Data Collection System. Am J Emerg Med 1988; 6:479–515.
9. Minnesota Department of Health, Minnesota Center for Health Statistics: Poison Statistics, Minnesota. Minneapolis, Minnesota, 1987.
10. US Department of Agriculture: Crop and Livestock Pesticide Usage Survey. Economic Research Service, 10S-2, 7–12. Washington, DC: Govt Printing Office, 1983.
11. Marquis JK: Contemporary Issues in Pesticide Toxicology and Pharmacology. Basel, New York: Karger, 1986.
12. Minnesota Agricultural Statistics Service: Pesticides used on Minnesota Farms. St. Paul, Minnesota, 1984.
13. Wasserstrom RF, Wiles R: Field duty: US farmworkers and pesticide safety. Washington, DC: World Resources Institute, 1985.
14. Bureau of National Affairs, Inc: Chemical Regulation Reporter 1990; 0148–7973/90:1289.
15. Gold E, Gordis L, Tonascia J, Szklo M: Risk factors for brain tumors in children. Am J Epidemiol 1979; 109:309–319.
16. Infante PF, Epstein SS, Newton WA Jr: Blood dyscrasias and childhood tumors and exposure to chlordane and heptachlor. Scand J Work, Enviorn Health 1978; 4:137–150.
17. Blanc PD, Olson KR: Occupationally related illness reported to a regional poison control center. Am J Public Health 1986; 76:1303–1307.
18. Rutstein DD, Brenberg W, Chalmers TC, Child III CG, Fishman AP, Perrin EB: Measuring the quality of medical care. N Engl J Med 1976; 294:582–588.
19. Rutstein DD, Mullan RJ, Frazier TM, Halperin WE, Melius JM, Sestito JP: Sentinel health events (occupational): A basis for physician recognition and public health surveillance. Am J Public Health 1988; 73:1054–1062.
20. Blanc PD, Rempel D, Maizlish N, Hiatt P, Olson KR: Occupational illness: Case detection by poison control surveillance. Ann Intern Med 1989; 111:238–244.

Lead Exposure in Outdoor Firearm Instructors

Ram K. Tripathi, PhD, Peter C. Sherertz, PhD, Gerald C. Llewellyn, PhD, and Carl W. Armstrong, MD

ABSTRACT

This study was conducted to determine lead exposure of firearm instructors at an outdoor firing range, while cadets were firing nonjacketed and jacketed lead ammunitions. The breathing zone air for lead exceeded the Occupational Safety and Health Administration standard of 50 $\mu g/m^3$ for two instructors during firing exercises using nonjacketed bullets. The use of totally copper-jacketed bullets reduced the breathing zone lead levels by 92 percent for instructor #1 and by 96 percent for instructor #2; subsequent blood lead levels showed a significant decline in both instructors. (*Am J Public Health.* 1991;81:753–755)

Introduction

Lead poisoning from exposure to airborne lead in indoor firing ranges is an occupational disease of public health concern. Several studies of occupational lead toxicity have been documented at indoor firing ranges.[1–7] The major route of absorption for lead in firing ranges is through inhalation of lead dust and fumes. Inadequate ventilation and lack of personal protective equipment use by instructors may result in lead toxicity.

Instructors assigned to firing ranges are likely to have higher exposures because they may spend significant time in that environment. Several occupational studies have suggested that exposure to lead may be a health risk for users of indoor firing ranges.[8–10] However, the extent of this risk among firearm instructors at outdoor firing ranges has not been documented. This study had two objectives: to evaluate the health risks to instructors from airborne lead exposure from nonjacketed, lead bullets; and to document the reduction or elimination of this risk by using totally copper-jacketed lead ammunition.

Methods

Two instructors, not involved in firing, were studied from June 17 to Septem-

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TABLE 1—Personal Breathing Zone Air Lead Levels of Instructors using Nonjacketed and Jacketed Lead Ammunition, 8-Hour, Time-weighted Averages, Richmond, 1987

Dates	Personal Air Lead Level ($\mu g/m^3$)	
	Instructor #1	Instructor #2
Nonjacketed bullets		
June 18, 1987	36.7	49.1
June 19, 1987	95.6	431.5
July 7, 1987	69.0	152.6
Mean	67.1	211.1
Copper-jacketed bullets		
September 4, 1987	5.4	8.7
Reduction	92.0%	96.0%

TABLE 2—Blood Lead Levels of Instructors using Nonjackoted and Jacketed Lead Ammunition, Richmond, 1987[*]

Dates	Blood Lead Level (µmol/L)	
	Instructor #1	Instructor #2
Nonjacketed bullets		
June 18, 1987	1.01	0.48
June 19, 1987	1.30	0.77
July 7, 1987	1.21	0.77
Mean	1.16	0.68
Copper-jacketed bullets		
September 4, 1987	1.06	0.63

[*] Pre-exposure baseline blood lead levels collected on June 17, 1987, in µmol/L, for instructors 1 and 2 were 1.01 and 0.48, respectively.

ber 4, 1987. Cadets fired a total of 950, 1,539, 3,000 nonjacketed, and 2,160 jacketed bullets on June 18, 19, July 7, and September 4, 1987, respectively. The total number of cadets involved in firing were seven, seven, six, and six on June 18, 19, July 7, and September 4, 1987, respectively. A description of the covered outdoor range was reported previously.[11] Thirty-eight caliber police revolvers and conventional, nonjacketed lead bullets (.38 special caliber, manufactured by 3D Inv, Inc., Doniphan, NE) were used, as well as totally copper-jacketed lead bullets (.38 caliber special ammunition, Omark Industries, Lewiston, IA).

All background, general area, and breathing zone air samples were collected on cellulose ester filters following standard procedures.[11] Background and general area samples were collected within the perimeter of the range one hour before and during firing, respectively. The breathing zone samples were collected near the chest and face of the instructors. All samples were analyzed by atomic absorption spectrophotometry.[12]

Prior to firing activities, individual interviews were conducted and questionnaires administered to obtain information on demographic and other potential sources of lead exposure. Venous blood was drawn to obtain baseline values for blood lead on June 17, 1987. A second questionnaire was administered on July 7, 1987 to assess any symptoms of lead toxicity and to ascertain any other recent potential sources of lead exposure. A third questionnaire was administered on September 4, 1987 after the study was completed. Blood was drawn on June 18, 19, July 7, and September 4, 1987 after each firing activity. All samples were analyzed according to the method of Brodie and Routh (1984)[13] by the National Health Laboratory, Vienna, VA.

Results

The mean concentration of lead in $\mu g/m^3$ for June 18, 19 and July 7, 1987 was 0.8 (range 0.3–1.2) in the background area air samples and 87 (range 3.8–299) in general area air samples during the firing. The mean concentrations of lead for background and general area air samples during use of copper-jacketed bullets for September 4, 1987 were 0.5 $\mu g/m^3$ and 9.5 $\mu g/m^3$, respectively, as previously reported.[14] Use of copper-jacketed ammunition resulted in an 89 percent reduction in lead levels in general area air samples.

The instructors were exposed to different personal breathing zone air lead concentrations on two sampling dates (Table 1). The mean breathing zone lead level with nonjacketed bullets was 67.1 $\mu g/m^3$ for instructor #1, and 211.1 $\mu g/m^3$ for instructor #2. The mean breathing zone lead levels during use of the jacketed bullets for instructors 1 and 2 were 5.4 $\mu g/m^3$ and 8.7 $\mu g/m^3$, respectively. This resulted in a reduction of 92 percent for instructor #1 and 96 percent for instructor #2.

After use of nonjacketed lead bullets, the mean blood lead level was 1.16 µmol/L for instructor #1, and 0.68 µmol/L for instructor #2 (Table 2). None of these values exceeded the current Occupational Safety and Health Administration (OSHA) return standard of 1.93 µmol/L (40 µg/dl) or removal standard of 2.4 µmol/L (50 µg/dl).[15] After the use of copper-jacketed lead bullets, blood lead levels for instructors 1 and 2 were 1.06 µmol/L and 0.63 µmol/L, respectively.

Discussion

All personal breathing zone lead level samples collected on June 19 and July 7 were above the current OSHA standard of 50 $\mu g/m^3$.[15] The majority of air lead levels for general area air samples and all breathing zone samples taken while using nonjacketed lead bullets were above the action level of 30 $\mu g/m^3$, for an eight-hour, time-weighted average (TWA).

The instructor blood lead levels measured during the nonjacketed lead bullet study indicated increases in blood lead concentrations. Blood lead levels reflect very recent lead exposures and are influenced by inhalation and ingestion. They may not reflect the total body burden of lead. Several studies have suggested that inhalation of air containing 1 $\mu g/m^3$ will increase the blood lead concentrations of an adult by about 0.048 µmol/L when air lead concentrations range from 1 to 5 $\mu g/m^3$.[16–18] Public health officials, as well as range owners, should be aware of design problems and work habits in outdoor ranges, especially covered ranges where exposures of instructors and other users may occur.

While no definite case of lead toxicity was encountered herein, the instructors experienced increased levels of lead in their blood while cadets were firing nonjacketed lead bullets. Interviews conducted with instructors revealed that the firearm instructors commonly supervise new cadets two to three times per month. This intermittent supervision probably has prevented serious lead toxicity in these instructors. If other covered outdoor firing ranges have air lead levels similar to those found in this study, frequent users would have an increased health risk from the lead exposure based on the present data.

The present study emphasizes a potential health risk of exposure to lead for firearm instructors in this type of outdoor firing range. Such levels were attributed to the firing of conventional, nonjacketed, lead bullets by the cadets. Inadequate ventilation may have contributed to the elevated lead levels found in the general area air samples and personal samples of the instructors. This potential health risk was substantially reduced by using totally copper-jacketed ammunition. The use of jacketed ammunition, as shown in this study and other studies, was very effective in reducing the air lead levels in general area and personal breathing zone air samples.[19–22] Even with such short-time exposures, instructors showed elevated blood lead levels. Remedies to such exposures not only include the use of jacketed ammunition but the adequate ventilation of the facility. □

Acknowledgments

The authors gratefully acknowledge the assistance of S.L. Ramsey for collecting blood samples; the Virginia State Police Academy for allowing us to conduct the study; the instructors who participated in this study; and Barbara Spain for typing the manuscript. Appreciation is extended to Grayson B. Miller, Jr, MD, Office of Epidemiology, Virginia Department of Health, as well as the Department of General Services, Virginia Division of Consolidated Laboratory Services for the assistance provided. The views expressed in this paper are those of the authors, and do not necessarily express any official position or policy relevant to the authors' affiliation. This paper was presented, in part, at the 29th Annual Meeting of the Society of Toxicology, February 12–16, 1990, Miami Beach, FL.

References

1. Anderson KE, Fischbein A, Kestenbaum D, Sassas S, Alveres AP, Kappas A: Plumbism from airborne lead in a firing range: An unusual exposure to a toxic heavy metal. Am J Med 1977; 63:306–312.
2. Valway SE, Martiny JW, Miller JR, Cook M, Mangione ET: Lead absorption in indoor firing ranges. Am J Public Health 1989; 79:1029–1032.
3. Olmez I, Kolra JP, Lowery S, Zoller WH: Airborne lead and trace elements in an indoor shooting range: A study of the DC National Guard Armory Pistol Range. Environ Toxicol Chem 1985; 4:447–452.
4. Dams R, Vandecasteele C, Desmet B, *et al:* Element concentrations in the air of an indoor shooting range. Sci Total Environ 1988; 77:1–13.
5. Novotony T, Cook M, Hughes J, Lee SA: Lead exposure in a firing range. Am J Public Health 1987; 77:1225–1226.
6. Anania TL, Seta JA: Lead exposure and design considerations for indoor firing ranges. NIOSH Technical Information. HEW Pub. No. (NIOSH) 76-130, Washington, DC: Govt Printing Office, 1975.
7. Anania TL, Lucas JB, Seta JA: Lead exposure in an indoor firing range. NIOSH Survey, Division of Technical Services, HEW Pub. No. (NIOSH) 74-100. Washington, DC: Govt Printing Office, 1975.
8. Fischbein A, Rice C, Sakozi L, Kon SH, Petrocci M, Selikoff IJ: Exposure to lead in firing ranges. JAMA 1979; 241:1141–1144.
9. Landrigan PJ, McKinney AS, Hopkins LC, Rhodes WW, Price WA, Cox DH: Chronic lead absorption: Result of poor ventilation in an indoor pistol range. JAMA 1975; 234:394–397.
10. Smith DL: Lead absorption in police small-arms instructors. J Soc Occup Med 1976; 26:139–140.
11. Tripathi RK, Sherertz PC, Llewellyn GC, Armstrong CW, Ramsey SL: Overexposure to lead at a covered outdoor firing range. J Am Coll Toxicol 1989; 8:111–117.
12. National Institute for Occupational Safety and Health: NIOSH Manual of Analytical Methods, 2nd Ed. P & CAM No. 173. Cincinnati, Ohio, DHEW Pub. No. (NIOSH) 1977; 77–157A.
13. Brodie KG, Routh MW: Trace analysis of lead in blood, aluminum and manganese in serum and chromium in urine by graphite furnace atomic absorption spectrometry. Clin Biochem 1984; 17:19–26.
14. Tripathi RK, Sherertz PC, Llewellyn GC, Armstrong CW, Ramsey SL: Reducing exposures to airborne lead in a covered, outdoor firing range by using totally copper-jacketed bullets. Am Ind Hyg Assoc J 1990; 51:28–31.
15. Occupational Safety and Health Administration: Occupational exposure to lead-final standard. Federal Register 1978; 43:52952–54616.
16. World Health Organization: Environmental Health Criteria 3. Lead. Geneva: WHO, 1977.
17. US Environmental Protection Agency: Air Quality Criteria for Lead, vol 1 of 4. EPA 600/8-83/028aF. Research Triangle Park, NC: USEPA Office of Research and Development, June 1986.
18. Carson BL, Ellis HV, McCann JL: Toxicology and biological monitoring of metals in humans; including feasibility and need. Chelsea, MI: Lewis Publishers, 1986.
19. Fischbein A, Nicholson WJ, Weisman I: Comparative lead emissions from conventional and jacketed ammunition. Am Ind Hyg Assoc J 1980; 41:525–527.
20. National Bureau of Standards: The reduction of airborne lead in indoor firing ranges by using modified ammunition. National Bureau of Standards Special Pub. 480-26. Washington, DC: US Department of Commerce, 1977.
21. Centers for Disease Control: Reducing exposure to airborne lead in indoor firing ranges—United States. MMWR 1983; 32:483–489.
22. Lee SA: Reducing airborne lead exposure in indoor firing ranges. FBI Law Enforcement Bulletin, February 1986; 15–18.

Soft Plastic Bread Packaging: Lead Content and Reuse by Families

Clifford Weisel, PhD, Michele Demak, MPH, Steven Marcus, MD, and Bernard D. Goldstein, MD

ABSTRACT

The presence of lead in labels painted on soft plastic bread packaging was evaluated. Lead was detected on the outside of 17 of 18 soft plastic bread bags that were analyzed, with an average of 26 ± .6 mg per bag with lead. Of 106 families questioned, 16 percent of respondents reported turning the bags inside out before reusing for food storage, thus putting food in contact with the lead paint. We estimate that a weak acid, such as vinegar, could readily leach 100 μg of lead from a painted plastic bag within 10 minutes. Further, lead and other metals painted on food packaging of any type becomes part of the municipal waste stream subject to incineration and to landfilling. The use of lead in packaging presents an unnecessary risk to public health. (*Am J Public Health.* 1991; 81:756–758)

Introduction

Low level effects of lead on intelligence and on behavior have been repetitively documented,[1-6] and a causal relation between low levels of lead and fetal malformations, adult hypertension, and auditory acuity has been suggested.[7-10] This recognition has led to efforts to diminish lead exposure, particularly from older homes containing indoor lead paint and from gasoline.

Lead and other metals are also present in colored plastics. However, only submicrogram/cm^2 amounts were extractable in acid or ethanol solutions when these are imbedded in the plastic itself.[11,12] Pigments used to decorate plastics sometimes included lead chromates[13] and if used on food wrappings the potential for transference to food exists.[14,15] While such painting is done on the outside of plastic food wrappings, the opportunity exists for the paint to come in contact with food, particularly if the soft plastic bag is reused. Additionally, the wrappings eventually are included in municipal solid waste disposal facilities and, therefore, the added lead is transferred to the environment.

Methods

Lead Analysis

We purchased 18 breads representing 14 different national brands from three different national chain supermarkets in three New Jersey towns about 80 miles apart: Westfield; Highland Park; and Woodbury. The breads included: white bread, whole wheat, bagels, and pita. A 50 or 200 cm^2 section of each wrapper, containing the printing, was removed, weighed, rinsed with distilled water, and placed in an acid washed beaker. Sections of clear plastic were also cut from several bags to serve as blanks. The total lead content was determined by either a hot or cold concentrated acid extraction. The hot acid extract consisted of 5 mL of nitric acid followed by heating at 250 °C until approximately 0.5 mL remained, then 5 mL HCl with heating until 0.5 mL remain. The cold acid extraction used 5 mL of HCl. The residue from each treatment was diluted to 25 mL with distilled water and analyzed using flame atomic absorption spectroscopy.

Survey Methods

A survey was designed to assess the extent to which soft plastic bread packages may be reused. We targeted families with young children, the subgroup of major concern for increased lead body burden. Two parent groups were surveyed by questionnaire in May and June of 1990. Fifty-three of 84 questionnaires were returned from the first group, and 53 of 90 from the second group (overall response rate 61 percent).

Most respondents are in their thirties, and are well-educated mothers of young children. They are the usual buyers of bread for their households, buying an average of two such packages per week.

Results

Lead was detected in the sections containing printing or designs on all but one of the soft plastic bread bags that were analyzed, but was not present in any of the clear sections of the bags (Table 1). The mean ± S.E. amount of lead for these bags with lead was 26 ± 6 mg. Incubation of the plastic wrappers in 5 percent acetic acid resulted in extraction of 6.8 percent of the lead pigment (mean of 4 studies) in 10 minutes and 10 percent in 60 minutes (mean of 6 studies).

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Editor's Note: See also related editorial on page 685 of this issue.

TABLE 1—Amount of Lead in Plastic Food Wrappers from Fourteen Brands of Bread (concentrated acid extraction)

Bread	Amount per painted portion (ug/cm²)	Estimated % of bag with painted writing	Total Lead per bag (mg)
White	7.4	90	13
White	7.8, 27	60	9.4, 32
White	46, 36	80	58, 74
White	14	40	11
White	2, 7.3, 44	95	3.8, 14, 84
White	17	80	27
Pita	17	50	9
Italian	ND	50	ND
Rye	0.9	50	0.9
White	7.9	40	6.3
White	22	95	39
Pita	2.8	50	1.1
White	29	75	44
Rolls	13	40	10

ND—not detected.

An average bag size of 2000 cm² was used to calculate the total lead content on the bags of white bread, 1000 cm² for the smaller bags.

Survey Results

Of the 106 respondents, 41 (39 percent) report reusing the packaging or bag from bread products. The reasons for doing so included: concern for the environment (34 families); convenience (24 families); and saving money (17 families). Food storage and/or packing lunch were the uses most often cited, with more than half of the "reusers" reporting these applications. These packages may be used on multiple occasions: eight families reused them at least three times.

Of the 41 reusers, 23 do so after turning the package inside out, and 17 of these families report using the bags while inside out for food storage. Thus 17 of the 106 respondents (16 percent) store food in contact with the painted label.

Preliminary analyses indicate no relationship between respondents' level of education and package reuse, although respondents who attended graduate or professional school are somewhat more likely to turn the bags inside out. Similarly, no obvious or significant relationship emerges for respondents who are full-time homemakers compared with those working outside the home. Anecdotal information suggests that the rationale for turning the bag inside out before reuse includes removal of bread crumbs that might become moldy, and drying the inside of the bag which picked up moisture from the bread. We were told of paint flaking from the inverted package onto stored food.

Discussion

The Environmental Protection Agency (EPA) estimates that 10 percent of municipal waste is incinerated, 10 percent recycled, and the remainder ends up in landfill.[16] The Food and Drug Administration (FDA) estimates that between 35 and 55 g of white bread and an additional 15 to 25 g of rolls and whole wheat bread are consumed daily across several age groups.[17] Assuming that each package of bread contains one pound and that 60 g of bread are consumed daily, 3.0×10^7 bread bags are disposed daily in the US. Assuming bread bags have an average lead content of 26 mg, 0.8 metric tons of lead would be added to the municipal waste stream daily. This lead could enter the environment as a result of incineration or leaching into groundwater from a landfill. Plastics are currently a prime candidate for recycling. The reduction of toxins is needed[16] to minimize exposure to workers involved in recycling and to increase potential uses of the final product.

EPA estimates that the daily baseline ingestion of lead from food, water, and beverages is between 32 and 40 μg.[17] Inhalation is estimated to give an additional exposure of 20 μg daily.[18] Based on our present findings, we conservatively estimate that a weak acid, such as vinegar, in contact with the pigment for as little as 10 minutes, could extract about 5 percent of the lead. For a 100 cm² surface, the size of a single slice of bread, this would produce an exposure of about 100 μg of lead.

Efforts are underway to reduce lead exposures through the removal of lead from gasoline, solder from cans, and, most importantly, from homes with deteriorated lead paints. However, our findings suggest that an additional source that is dependent upon the habits of individuals, and not measured by current procedures for food analysis, may need to be considered when estimating total lead exposure.

Although presenting an unnecessary risk to health which should be discontinued, we do not believe it likely that the use of inverted bread packaging to store or transport food presents an immediate threat to the health of family members necessitating an emergency response. However, obtaining blood free erythrocyte protoporphyrin or lead levels is a valuable part of the routine medical evaluation of all young children and is particularly important for high-risk groups.[18] Until this use of lead is prohibited, plastic packaging should not be turned inside out to store or transport food. ☐

Acknowledgments

We acknowledge the assistance of Arlene Yang, Ryun Yu, Elaine Randolph, and Eugene F. Peters, Jr. Supported by NIEHS Center Grant #ES05022.

References

1. Needleman HL: The persistent threat of lead: A singular opportunity. Am J Public Health 1989; 79:643–645.
2. Davis JM, Svendsgaard DJ: Lead and child development. Nature 1987; 329:297–300.
3. Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M: Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. N Engl J Med 1987; 316:1037–1043.
4. McMichael AJ, Baghurst PA, Wigg NR, Vimpani GV, Robertson EF, Roberts RJ: Port Pirie cohort study: Environmental exposure to lead and childrens abilities at the age of four years. N Engl J Med 1988; 319:468–475.
5. US Department of Health & Human Services, Agency for Toxic Substances & Disease Registry: Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. Washington, DC: DHHS, July 1988.
6. Fulton M, Thompson G, Hunter R, Raab G, Laxen D, Hepburn W: Influence of blood lead on the ability and attainment of children in Edinburgh. Lancet 1987; 10:1221–1226.
7. Schwartz J, Otto DA: Blood lead, hearing threshold and neurobehavioral development in children and youth. Arch Environ Health 1987; 42:153–160.
8. Mushak P, Davis JM, Crocetti AF, Grant LD: Prenatal and postnatal effects of low-level lead exposure: Integrated summary of

a report to the US Congress on childhood lead poisoning. Environ Res 1989; 50:11–36.

9. Pirkle JL, Schwartz J, Landis JR, Harlan WR: The relationship between blood lead levels and blood-pressure and its cardiovascular risk implications. Am J Epidemiol 1985; 121:246–258.
10. Sharp DS, Smith AH, Holman BL, Fisher JM, Osterloh J, Becker CE: Elevated blood pressure in treated hypertensives with low-level lead accumulation. Arch Environ Health 1989; 44:18–22.
11. Meranger JC, Cunningham HM, Giraoux A: Extraction of heavy metals from plastic food containers: An x-ray fluorescence and atomic absorption study. Can J Public Health 1974; 65:292–295.
12. Alam MS, Srivastava SP, Seth PK: Factors influencing the leaching of heavy metals from plastic materials used in the packaging of food and biomedical devices. Indian J Environ Health 1988; 30:131–141.
13. Mimeault VJ: Colorants for plastics. Additives for Plastics, Vol. 2. New York: Academic Press, 1978.
14. Reilly C: Metal Contamination of Food. London: Applied Science Publishers, 1980.
15. Preda N, Popa L, Ariesan M: The possibility of food contamination with cadmium by means of coloured plastics. J Appl Toxicol 1983; 3(3):139–142.
16. US Congress, Office of Technology Assessment: Facing America's Trash: What Next for Municipal Solid Waste? Washington, DC: OTA-424, 1989.
17. US Environmental Protection Agency: Air Quality Criteria for Lead Vol. II. EPA 600/8-83/028 BF. Washington, DC: EPA, June 1986.
18. American Academy of Pediatrics, Committee on Environmental Hazards, Committee on Accident and Poison Prevention: Statement on childhood lead poisoning. Pediatrics 1987; 79:457–465.

Non-Hodgkin's Lymphoma in a Cohort of Vietnam Veterans

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ABSTRACT

We examined the incidence of non-Hodgkin's lymphoma (NHL) in a cohort of 18,313 United States Army veterans from the Vietnam era. Diagnoses were confirmed through a review of hospital records. Among veterans who had died after discharge or who had participated in a telephone interview (8,170 Vietnam veterans and 7,564 non-Vietnam veterans), seven Vietnam veterans and one non-Vietnam veteran had developed non-Hodgkin's lymphoma ($p = 0.07$). As none of the NHL cases had military job titles which suggest that they were occupationally exposed to herbicides while in Vietnam, the reasons for the excess are unclear. (*Am J Public Health.* 1991;81:758–760)

Introduction

Results of studies of occupational groups exposed to phenoxy herbicides have raised concerns that Vietnam veterans may have an increased risk of developing non-Hodgkin's lymphoma (NHL) because of possible exposure to the phenoxy herbicide Agent Orange, used in South Vietnam between 1966 and 1970.[1–3] These concerns have been heightened by reports of an increased risk of NHL among Vietnam veterans.[4,5] In the Centers for Disease Control Vietnam Experience Study (VES), the health of US Army Vietnam veterans was compared with that of Vietnam-era Army veterans who had not served in Vietnam. Although the VES was not designed to assess the risk of rare cancers among Vietnam veterans, the high level of concern about NHL led us to investigate and report the findings for NHL.

Methods

The study population consisted of 18,313 randomly selected, male US Army veterans (9,324 Vietnam veterans and 8,989 non-Vietnam veterans) who entered military service between January 1965 and December 1971. The VES included both mortality and health interview components, and the methods have been described in detail elsewhere.[6,7]

In this analysis, we combined mortality and interview data to determine the number of veterans with NHL in the cohort. Two hundred forty-six Vietnam veterans and 200 non-Vietnam veterans had died between separation from active duty and December 31, 1983, the closing date of the mortality component. A panel of physicians examined medical records of these veterans to determine the causes of death for each veteran.

In the interview component of the VES, we attempted to locate and contact all veterans not identified as deceased. Altogether, we interviewed 15,288 veterans (87.3 percent of eligible Vietnam veterans and 83.8 percent of non-Vietnam veterans) by telephone during 1985–86. If the veteran reported that he had been diagnosed with cancer, we asked him to describe the type of cancer that he had. To verify three self-reported cases of NHL and to identify other cases, we sought medical records for 47 veterans, including men who reported a malignant neoplasm of the lymphatic or hematopoietic tissues, men who reported a diagnosis of cancer of a site that could be NHL (e.g. lung, neck, stomach, lymph nodes), and men who reported a diagnosis of cancer but could not name a specific type or site. We could not

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From the Center for Environmental Health and Injury Control, Centers for Disease Control. Address reprint requests to Thomas R. O'Brien, MD, MPH, CDC, 1600 Clifton Road (E45), Atlanta, GA 30333. This paper, submitted to the Journal May 25, 1990, was revised and accepted for publication January 23, 1991.

TABLE 1—Source, Diagnosis, Age at Diagnosis, Race, Period of Vietnam Service, Military Occupation, Military Region of Service, and Latency Period of Vietnam Veterans Diagnosed with Non-Hodgkin's Lymphoma, Vietnam Experience Study 1985–86

Source	Diagnosis[a]	Age at Diagnosis	Race	Vietnam Service	Military Occupation	Military Region	Months of "Latency"[b]
Interview	Diffuse mixed lymphocytic-histiocytic lymphoma	35	White	1968–69	Infantryman	III	172
Interview	Burkitt's lymphoma[c]	34	White	1968–69	Clerk Typist	III	148
interview	Poorly differentiated lymphocytic lymphoma	25	White	1968–70	Supply	I & III	75
Interview	Malignant lymphoma, large cell type	37	White	1966–67	Radar Maintenance	III	211
Mortality	Burkitt's lymphoma[d]	34	White	1968–69	Artilleryman	II & III	175
Mortality	Lymphoblastic lymphoma	24	White	1968–69	Infantryman	II	47
Mortality	Reticulum cell sarcoma	26	White	1968–69	Administrative Personnel Specialist	III	8

[a] On the basis of medical records.

[b] The "latent period" for NHL was taken as the time period between the beginning of the veteran's tour of duty in Vietnam and the initial medical contact that led to the diagnosis of NHL.

[c] On the basis of review of microscopic tissue specimen, diagnosis was malignant lymphoma (diffuse, small non-cleaved cell type) or Burkitt's lymphoma.

[d] On the basis of review of microscopic tissue specimen, diagnosis was poorly differentiated lymphoid malignancy with extensive sclerosis, consistent with acute lymphoblastic leukemia.

obtain medical records for six of the 47 veterans; none of the six had reported a lymphatic or hematopoietic cancer during the interviews. We made final diagnoses on the basis of pathology reports found in the medical records or, when no pathology report was available, the preponderance of evidence from the medical records. Pathology records were available in 31 of 41 (75.6 percent) of these medical records.

For analytic purposes, we divided the number of veterans with NHL (ascertained through either the mortality component or the interview) in each cohort by the sum of the number of veterans who had died and the number of veterans who had been interviewed (8,170 Vietnam veterans and 7,564 non-Vietnam veterans).

Results

We confirmed that all three veterans who had reported a history of NHL during the interview had been so diagnosed. A fourth veteran who, in the interview, had not specified the type of cancer had also been diagnosed with NHL. All four of these men were Vietnam veterans (Table 1). In the mortality study, three Vietnam veterans and one non-Vietnam veteran were found to have NHL. In total, seven Vietnam veterans and one non-Vietnam veteran were diagnosed with some type of NHL ($p = 0.07$, Fisher exact test (two-tailed)). On the basis of published age-specific cancer incidence data,[8] about 3.9 cases of NHL would have been expected among Vietnam veterans ($p = 0.10$, Poisson) and about 3.5 cases would have been expected among non-Vietnam veterans ($p = 0.14$, Poisson).

All of the Vietnam veterans with NHL had begun their tour in Vietnam from 1966 to 1968. The men had served in a variety of military occupations, and no specific occupation predominated. Regarding pathology, two Vietnam veterans were diagnosed as having Burkitt's lymphoma. On further pathologic review, one case was reported as possibly Burkitt's lymphoma, and the second was reported as acute lymphoblastic leukemia (Table 1).

The "latency period" was short for two Vietnam veterans (Table 1). One veteran first developed NHL eight months after arriving in Vietnam and a second Vietnam veteran had a "latency period" of 47 months.

Discussion

Although these results suggest an increased risk of NHL among Vietnam veterans, our findings should be interpreted with caution. The number of cases is small and, on review, one of the Vietnam veterans may have had lymphocytic leukemia rather than NHL. (These diseases are, however, closely related.[9]) The time period between the beginning of the veteran's tour of duty in Vietnam and the diagnosis of NHL should also be considered when interpreting these data, as the "latent period" for some of the cases presented here is much shorter than the usual period for environmentally caused cancers.[10]

The collective evidence for an unusual risk of NHL among Vietnam veterans is inconsistent. Two other studies have shown that Vietnam veterans may be at increased risk of NHL. Increased mortality from NHL was seen in a large study of US Marine Corps Vietnam veterans (proportionate mortality ratio (PMR) = 2.10; 95% confidence interval = 1.17–3.79), although the same investigators found a deficit of deaths from NHL in US Army veterans (PMR = 0.81; 95% CI = 0.63–1.04).[4] An elevated risk of NHL in Vietnam veterans was also reported in a large population-based case-control study (Odds ratio = 1.47; 95% CI = 1.09–1.97).[5] In contrast, a case-control study using subjects from Veterans Administration hospitals found a modest negative association between Vietnam service and NHL (OR = 0.72; 95% CI = 0.56–0.92).[11]

We could only indirectly assess possible exposure of these veterans to Agent Orange while in Vietnam. Although most of the men served in Vietnam during the time (1967–69) and area (III Corps) of heaviest spraying,[1] three of seven had non-combat military job titles that suggest they were unlikely to have been in contact with herbicides. Although the three men with combat-related job titles may have been exposed to Agent Orange, previous work suggests that, with the exception of men whose military occupation entailed handling herbicides, most US Army combat troops who served in Vietnam were not heavily exposed to dioxin.[12] Since no other factor in the Vietnam experience has been linked to the increased NHL risk, reasons for the excess found in this study and two other studies remain unclear. ☐

Acknowledgments

Linda Moyer, RN, obtained and abstracted medical records that were reviewed in this study. Dr. Elaine S. Jaffe reviewed microscopic tissue specimens. Sandra Tully and Sheila Allgood assisted with manuscript preparation.

Data for this study were gathered under an interagency agreement with the Veterans Administration.

References

1. Committee on the Effects of Herbicides in Vietnam: The effects of herbicides in South Vietnam. Part A—Summary and conclusions. Washington, DC: National Academy of Sciences, 1974; 111–117.
2. Hardell L, Ericksson M, Lenner P, *et al:* Malignant lymphoma and exposure to chemicals, especially organic solvents, chlorophenols and phenoxy acids: A case-control study. Br J Cancer 1981; 43:169–176.
3. Hoar SK, Blair A, Holmes FF, *et al:* Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. JAMA 1986; 256:1141–1146.
4. Breslin P, Kang HK, Lee Y, Burt V, Shepard BM: Proportionate mortality study of US Army and US Marine Corps veterans of the Vietnam war. JOM 1988; 30:412–419.
5. Selected Cancers Cooperative Study Group. The association of selected cancers with service in the US military in Vietnam. I. Non-Hodgkin's Lymphoma. Arch Intern Med 1990; 150:2473–2483.
6. Centers for Disease Control Vietnam Experience Study. Postservice mortality among Vietnam veterans. JAMA 1987; 257:790–795.
7. Centers for Disease Control Vietnam Experience Study: Health status of Vietnam veterans. I. Psychosocial characteristics. JAMA 1988; 259:2701–2707.
8. National Cancer Institute: Surveillance, epidemiology, and end results: Incidence and mortality data, 1973–77. National Cancer Institute Monograph 57. NIH Pub. No. 81-2330. Washington, DC: Govt Printing Office, 1981.
9. Heath CW Jr: The leukemias. *In:* Schottenfeld D, Fraumeni JF Jr (eds): Cancer Epidemiology and Prevention. Philadelphia: WB Saunders, 1982; 728–738.
10. Decouflé P: Occupation. *In:* Schottenfeld D, Fraumeni JF Jr (eds): Cancer Epidemiology and Prevention. Philadelphia: WB Saunders, 1982; 318–335.
11. Dalager N, Kang H, Burt V: Non-Hodgkin's lymphoma among Vietnam-era veterans (abstract). Am J Epidemiol 1989; 130:815.
12. Centers for Disease Control Veterans Health Studies: Serum 2,3,7,8-tetrachlorodibenzo-p-dioxin levels in US Army Vietnam-era veterans. JAMA 1988; 260:1249–1254.

Endemic Giardiasis and Municipal Water Supply

G. Graham Fraser, MB, ChB, MSc, and Kenneth R. Cooke, MB, ChB, PhD

ABSTRACT

To test the hypothesis that endemic giardiasis may be transmitted by unfiltered municipal water supplies, the incidence of laboratory-confirmed giardiasis was studied in a natural experiment due to the arrangement of the public water supply of Dunedin, New Zealand. The incidence rate ratio was 3.3 (90% CI = 1.1, 10.1) for the population receiving unfiltered (microstrained) water relative to that using sand filtered water. In a parallel case-control study of incident cases, the odds ratio for giardiasis and unfiltered (microstrained) water supply was 1.8 (90% CI = 0.5, 6.9). **(*Am J Public Health.* 1991; 81:760–762)**

Introduction

Epidemics of waterborne giardiasis originating from water supplies have been clearly associated with surface catchments, treatment systems that do not eliminate *Giardia* cysts, and downstream contamination of the reticulation system.[1–5] In contrast, there have been few tests of the hypothesis that surface catchment waters contribute to endemic giardiasis where treatment systems cannot eliminate cysts.[1,6,7]

Giardia cysts have only recently been isolated from water supplies in New Zealand. No substantive epidemics attributable to giardiasis have yet been documented. Many municipal supplies use surface waters and simple treatment methods that would not reliably filter or deactivate giardia cysts.[3,8]

The present study took advantage of a natural experiment—the divided water supply of the city of Dunedin, New Zealand—to test the hypothesis that endemic giardiasis might be transmitted by unfiltered municipal water supplies.

Methods

Dunedin (population 89,000) is supplied almost entirely by water from surface catchments. Most water is filtered by mechanical microstrainers (screen size 23 μm). Part of the city water is treated at a modern station (Mount Grand) using coagulation/flocculation and direct dual media filtration (anthracite and silica sand) that would normally be expected to remove any *Giardia* cysts present.[3,8] All water supplies are chlorinated and fluoridated.

Records of all laboratory-proven cases of giardiasis from persons residing within the Dunedin municipal water sup-

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TABLE 1—Incidence of Giardiasis in Dunedin: Laboratory-Confirmed Primary Household Cases by Age and Sex*

Age (years)	Cases			Incidence Rate**
	Male	Female	Total	
0–4	5	3	8	7.8
5–14	0	2	2	0.9
15–34	11	15	26	4.3
35–64	5	5	10	2.1
65+	0	1	1	0.5
Total	21	26	47	2.9

*Three cases excluded from this table, as age or sex unspecified.
**Rate per 10,000 person-years.

TABLE 2—Incidence of Laboratory-Confirmed Giardiasis by Water Supply Filtration Method

Filtration Method	Cases*	Incidence**	Relative Risk (90% CI)
Direct dual media granular filtration	2	0.99	1.0 (reference group)
Microstrained	47	3.29	3.3 (1.1, 10.1)

*Primary household cases only. One case could not be classified, as domicile received a blend of both kinds of supply.
**Rate per 10,000 person-years.

TABLE 3—Giardiasis and Exposure to Microstrained Water: Case-Control Study

Exposed to Microstrained Water	Cases	Controls
Ever	19	53
Never	2	10
Odds Ratio (90% CI)	1.8 (0.5, 6.5)	

ply area were obtained from the two medical laboratories in the city for two periods (February to December 1986 and April 1987 to February 1988). Each record included age, sex, residential address, presence of *Giardia* or other pathogens, and the date of receipt of the fecal specimen. The laboratory diagnosis was made by direct microscopy following formalin-ethyl-acetate sedimentation and iodine staining. The denominators for rates were obtained from the 1986 Census of the usually resident populations of the respective water supply areas.[9]

A case-control study was also conducted using cases in the 1987–88 period. Controls were selected from the population referred for laboratory parasitology examination who had negative findings for giardiasis. Three controls were selected per case; cases and controls were matched for age, sex, and season of occurrence of infection. Cases and controls were classified blindly by city council water engineers according to whether their residences were "ever" exposed to microstrained water during the presumed period of infection (defined as seven days to four weeks before the receipt of the fecal specimen).[10]

Relative risks, odds ratios, and confidence limits were computed using the programs of Rothman and Boice.[11]

Results

Fifty primary household cases of giardiasis were diagnosed in the two study periods. Subsequent cases in a household were excluded from the analysis (13 cases). Four further cases were excluded through insufficient or absent information on domicile.

The incidence of laboratory-confirmed giardiasis was greatest in preschool children and in the 15–34 age group (Table 1). The incidence of laboratory-proven giardiasis for residents of the two water supply areas are given in Table 2. In the first study period, 28 of the 29 cases were residents in areas supplied by microstrained water; the remaining case was in an area traditionally supplied by a blend of the two supplies. In the second study period, 19 cases resided in the microstrained areas and two cases in areas that received the Mount Grand supply. The relative risk of laboratory-confirmed giardiasis for residents of areas supplied by microstrained water was estimated at 3.3 (one-tailed $P = 0.04$). The risk ratio was essentially unchanged following indirect standardization for age.

In the case-control study the odds ratio for exposure to microstrained water was 1.8 (90% CI = 0.5, 6.5) (Table 3).

Discussion

The present findings suggest that endemic giardiasis can originate from the municipal water supply. Both the incidence estimates and the case-control analysis indicate a greater risk of laboratory-diagnosed giardiasis among residents of areas whose supply was treated only by mechanical filtration and chlorination.

The study was small and of correspondingly weak statistical power. As it was limited to the analysis of laboratory reports, information on some possible confounding factors was not available. However, some potential confounders are either unlikely to have had a significant effect, or would have operated as negative confounders. Age differences were not sufficient to account for differences in rates observed. The area with low rates included the most mobile young adult population. The two population areas may have differed in physician practice styles in the investigation of gastrointestinal symptoms, but this should have been accounted for by the case-control study. As most workplaces are supplied by Mount Grand water, misclassification from consumption of cold water at work would be expected to bias the relative risk toward unity. A small proportion of the controls may have had giardiasis (due to the intermittent nature of *Giardia* excretion) but this would bias the odds ratio toward unity.

The consistency of our findings with those of other case-control and descriptive studies[1,6,7] strongly suggests that transmission of endemic giardiasis can occur via municipal water supplies that do not use filtration technology capable of removing *Giardia* cysts. ☐

Acknowledgments

The authors thank Norman Fitzgerald, MD, and John Finlayson of the Medical Laboratory, Dunedin; Roly Hayes, BE, of the Dunedin City

Council; and James Reid, MB, of the University of Otago Medical School for their assistance with the study and review of the manuscript.

References

1. Craun GF: Surface water supplies and health. J Am Water Works Assoc 1988; 80:40–52.
2. Craun GF: Waterborne giardiasis in the United States 1965–1984. Lancet 1986; 2:513–514.
3. Jakubowski W: Purple burps and the filtration of drinking water supplies. (Editorial) Am J Public Health 1988; 78:139–143.
4. Kent GP, Greenspan JR, Herndon JL, *et al:* Epidemic giardiasis caused by a contaminated public water supply. Am J Public Health 1988; 78:139–143.
5. Jephcott AE, Begg NT, Baker IA: Outbreak of giardiasis associated with mains water in the United Kingdom. Lancet 1986; 2:730–732.
6. Chute CG, Smith RP, Baron JA: Risk factors for endemic giardiasis. Am J Public Health 1987; 77:585–587.
7. Birkhead G, Vogt RL: Epidemiological surveillance for endemic *Giardia lamblia* infection in Vermont. Am J Epidemiol 1989; 129:762–768.
8. Ongerth JE: Evaluation of treatment for removing *Giardia* cysts. J Am Water Works Assoc 1990; 82:85–96.
9. NZ Department of Statistics: 1986 New Zealand Census of population and dwellings, series B. Wellington: Department of Statistics, 1987.
10. Benenson AS (ed): Control of Communicable Disease in Man. Washington, DC: American Public Health Association, 1986.
11. Rothman K, Boice JD: Epidemiologic analysis with a programmable calculator. Boston: Epidemiology Resources, Inc, 1978.

The Microbiologic Quality of Drinking Water in North Carolina Migrant Labor Camps

Stephen Ciesielski, PhD, Thomas Handzel, BS, and Mark Sobsey, PhD

ABSTRACT

A two-year study of the microbiological quality of drinking water in 27 randomly selected North Carolina migrant labor camps yielded total and fecal coliform prevalences of 44 percent and 26 percent, respectively in 1988 and similar but higher prevalences in 1989. Preoccupancy testing by county sanitarians had found virtually no total coliform contamination. These findings suggest that a potential source of contamination existed and that current testing protocols which rely on preoccupancy testing may be inadequate. (*Am J Public Health.* 1991;81:762–764)

Introduction

Although migrant farm workers are known to have a high prevalence of enteric infections,[1–7] many of which are water-borne, the potability of drinking water used by migrants has received little attention. This report presents the results of a two-year study of the microbiological quality of drinking water in North Carolina migrant labor camps.

Methods

In 1988, a random sample of 21 migrant camps was selected from a pooled list of camps in eastern North Carolina. Water was also tested in six additional camps, which were part of a related study of the incidence of enteric parasites in migrant farm workers, and 10 control sites (nearby rural businesses and residences). In 1989, all 23 inhabited study camps from the previous year were retested, along with seven additional nonrandomly selected camps. In all camps in both years, water was tested by county health departments prior to occupancy, the results of which were obtained after field study was completed.

Water samples were collected from taps (without aerators) into sterile 1 liter sampling bottles, kept chilled and analyzed within 24 hours. Microbiological testing of 100 ml sample volumes for total and fecal coliforms and *E. coli* was done according to standard protocols using membrane filter methods.[8] Sites with initial positive tests were retested when possible.

Fisher's Exact Test (two-tailed) and Chi-square tests were employed in data analysis.

Results

1988 Results

The bacteriological results for water samples from the 27 camps and 10 control sites are summarized in Table 1; 12 (44 percent) were positive for total coliforms with counts ranging from 1–186 colony forming units (CFU). Eight of these sites were retested and all were positive again. Seven samples (26 percent) were also positive for fecal coliforms. Two of 10 samples were positive for *E. coli*. All 10 control sites were negative for total and fecal coliforms. The presence of latrines (as opposed to flush toilets) was associated with total coliform occurrence in water (p = 0.024; Fisher's Exact Test).

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TABLE 1—Results of 1988 Microbiologic Testing of Drinking Water from Migrant Labor Camps and Control Sites

Sample Source	# Samples	Analyte	# (%) Positive	Mean Bacterial Concentration	
				Geometric	Arithmetic
Migrant Camps	27	T.C.*	12(44)	8.6	16.0
	27	F.C.**	7(26)	0.92	2.65
	10	*E. coli****	2(20)	1.81	4.5
Control Sites	10	T.C.	0	0	0
	10	F.C.	0	0	0

*Total Coliforms.
**Fecal Coliforms.
***Testing for *E. coli* was only performed for 10 of 12 camps with positive coliform results.

TABLE 2—Results of 1989 Microbiologic Testing of Drinking Water from Migrant Labor Camps

Sample Source	# Samples	Analyte	# (%) Positive	Mean Bacterial Concentration	
				Geometric	Arithmetic
Migrant Camps	30	T.C.*	15(50)	12.0	31.93
	30	F.C.**	11(37)	2.19	6.18
	30	*E. coli*	4(13)	0.93	1.0

*Total Coliforms.
**Fecal Coliforms.

1989 Results

Fifteen of 30 camps (50 percent) had water samples positive for total coliforms, 11 (37 percent) for fecal coliforms, and four for *E. coli* (Table 2). Fifty-six percent of resampled camps had the same result as the previous year. In 1989, latrines were not significantly associated with coliforms, but there was an increasing prevalence of coliforms in water samples as a function of time of occupancy. When the results of the four successive samplings of water are grouped as two early samplings and two late samplings, the total coliform prevalence in the early and late groups is significantly different by Chi-square analysis ($p = 0.056$). Rainfall during the week previous to the last two sampling periods totaled 3.68 inches while totaling 0.78 inches for the week prior to the first two sampling periods.[9]

In 1988 only one and in 1989 none of the camps studied were positive for total coliforms on preoccupancy testing by local health departments.

Discussion

In the United States over one-half of the reported outbreaks of waterborne disease are due to fecally contaminated groundwater, most often resulting from overflow or seepage of on-site septic systems.[10] Migrant camps are often overcrowded so that the amount of human fecal wastes may overload the design capacity of on-site sanitary disposal systems. These septic systems may also have exceeded their expected life span. Furthermore, migrants probably excrete greater numbers of pathogens into the septic waste system. Enteric infections, especially parasites, are major health problems among migrants.[1–7] Diarrhea is reported to be 20 times more frequent among migrants than the urban poor.[4] Most intestinal parasites identified in the migrant population can be transmitted by fecally contaminated water[1,3,5,11] and enteric bacteria and viruses have a much greater capacity for waterborne transmission.[7] The concurrent study of the incidence of intestinal parasites in which some study camps were included, found a significant association ($p = .024$) between incidence and total coliforms.* In Florida an echovirus (type 22/23 complex) was isolated from water samples in a migrant camp during an outbreak of gastrointestinal illness.[12]

The surprisingly high prevalence of total and fecal coliforms in migrant labor camp drinking water is a matter of serious concern. According to the US Environmental Protection Agency, "the presence of fecal coliforms in drinking water indicates that an urgent public health problem exists."[14] New standards proposed by the EPA would require the state to be notified within 48 hours when any number of fecal coliforms are demonstrated.[13] The health hazards associated with unsafe drinking water in migrant camps were demonstrated in 1973, when the largest outbreak of typhoid fever in the US since 1939 resulted from the contamination of well water in a Florida migrant camp, preceded by repeatedly high coliform counts in the camp's water supply.[14]

North Carolina's migrant housing regulations require that water in migrant camps housing 13 or more workers be tested before occupancy for total coliforms.[15,16] The failure of pre-occupancy inspections to ensure adequate water quality during habitation probably results from the fact that if coliforms have entered the water system when the camp is occupied in late summer, results are negative the following June (after a nine-month vacancy) due to bacterial die-off and dilution and dispersion in ground water. This variation with usage is supported by the increase in coliform prevalence observed in 1989 with each round of sampling, which suggests that overload of on-site waste water treatment facilities may be a factor. Increasing rainfall in the early autumn may also play a role.

On the basis of these findings, it seems logical to suggest that current regulations be revised to include water quality testing both before and during the period of habitation, not only in North Carolina, but in all states in which migrants are served by ground water sources. Further study is needed to determine the factors responsible for the presence of coliforms in drinking water so that effective intervention can be initiated. □

Acknowledgments

Funding for this study was provided by the Rural Community Assistance Program, 602 South King Street, Suite 402, Leesburg, VA 22075; special thanks are expressed to Kathleen Stanley, Acting Director. Additional funding was provided by the Tri-County Community Health Center, Newton

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*Ciesielski SD, Seed JR, Ortiz JC, Metts J: The epidemiology of intestinal parasites among North Carolina migrant farmworkers: Results of a two-year long study. Unpublished data.

Grove, NC; special thanks are expressed to the clinic's Director, Michael Baker.

1. Ciesielski SD, Seed JR: Preliminary report: Intestinal parasites in adult migrant farmworkers in North Carolina. Migrant Health Newsline, Migrant Clinicians Network, Austin, Texas, 1988; 5:3–5.
2. Arbab DM, Weidner B: Infectious diseases and field water supply and sanitation among migrant farmworkers. Am J Public Health 1986; 76:694–695.
3. Ungar BL, Iscoe E, Cutler J, Bartlett JG: Intestinal parasites in a migrant farmworker population. Arch Intern Med 1986; 146:513–515.
4. Wilk VA: The Occupational Health of Migrant and Seasonal Farmworkers in the United States. Washington, DC: Farmworker Justice Fund, 1986.
5. Ortiz J: The prevalence of intestinal parasites in Puerto Rican farmworkers in western Massachusetts. Am J Public Health 1980; 70:1103–1105.
6. Centaur Associates, Inc: Final Report: Industry Profile Phase I. Study Field Sanitation. Washington, DC: Centaur Associates, September 1983.
7. Federal Register 52(84):16050–16096.
8. American Public Health Association: Standard Methods For the Examination of Water and Wastewater. Washington, DC: APHA, 1989.
9. National Climatic Data Center, Asheville, NC, September and October, 1988, and September and October, 1989.
10. Environmental Protection Agency: Septic Tank Siting to Minimize the Contamination of Ground Water by Microorganisms. Washington, DC: EPA, 1987.
11. Mcjunkin FE: Water and Human Health. Washington, DC: National Demonstration Water Project, 1982.
12. Wellings FM, Mountain CW, Lewis AL: Virus in groundwater. *In*: Proc. 2nd National Conference on Individual On-Site Wastewater Systems, Ann Arbor Science, Ann Arbor, MI 1977; 61.
13. Environmental Protection Agency: Proposed Rules. Federal Register, June 29, 1989, 54FR27544.
14. Hoffman TA: Waterborne typhoid fever in Dade County, Florida: Clinical and therapeutic evaluation of 105 bacteremic patients. Am J Med 1975; 59:481–487.
15. North Carolina Drinking Water Act, 130A-311.
16. North Carolina State and Federal Laws Affecting Migrant and Seasonal Farmworkers. Governors Special Grant, North Carolina Department of Natural Resources and Community Development, Raleigh, NC.

Salmonella Egg Survey in Hawaii: Evidence for Routine Bacterial Surveillance

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ABSTRACT

One hundred and six dozen eggs, representing 12 brands, were purchased from Oahu supermarkets and cultured for *Salmonella* using standard FDA (Food and Drug Administration) microbiological techniques. Two enrichment incubation temperatures were used to improve culture sensitivity. Ten cartons (9.4 percent) of the 106 dozen samples had shells positive for *Salmonella*. Seven of the 10 were traced to a single egg processor. Inspection of the facility led to the discovery of malfunctioning equipment used in the egg washing and sanitation process. (*Am J Public Health.* 1991;81:764–766)

Introduction

Large outbreaks of human salmonellosis in the United States, related to egg products and cracked shell eggs, led to the passage of the Egg Products Act in 1970. This law required visual inspection of eggs for cracks as well as mandating pasteurization for all bulk egg products.[1,2] Although this legislation led to an initial decrease in egg-related *Salmonella* outbreaks, they have again begun to rise.[2–5]

Contamination of eggs with *Salmonella* can occur in several ways: egg shells may be contaminated by feces[6]; cleaning procedures may be improper[7]; the interior of the egg may be contaminated by the shell being cracked. Contamination may also be internal, prior to shell formation through transovarian transmission.[5,8] In 1989, an Oahu high school student, as part of a science project, cultured two dozen eggs from a supermarket in Honolulu for *Salmonella*. Nine of 24 eggs (37 percent) were culture positive. The high recovery rate prompted the Hawaii State Department of Health to conduct a more definitive prevalence survey for *Salmonella* among eggs for sale in Oahu supermarkets.

Methods

Eggs representing 12 available brands were collected once each week for an eight-week period from June 26 through August 19, 1989. The sampling unit was defined as a carton of 12 large grade A eggs. The carton closest to the aisle was chosen without opening it. Following collection, the eggs were immediately transported to the Hawaii State De-

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TABLE 1—Culture Results of Egg Shells from Ten Dozen Eggs Positive for Salmonella Using Two Incubation Temperatures

Incubation Temp. 35 Degrees Celsius				Incubation Temp. 41 Degrees Celsius			
Dozen	Egg Brand	*Plating Medium	Serotype	Dozen	Egg Brand	*Plating Medium	Serotype
1	K	XLD	Braenderup	1	K	XLD	Braenderup
1	K	BGA	Braenderup	1	K	BGA	Braenderup
				2	K	XLD	Oranienburg
3	J	BGA	Oranienburg	3	J	BGA	Oranienburg
				3	J	XLD	Oranienburg
				4	H	BGA	Mbandaka
5	K	BGA	Cerro				
5	K	XLD	Cerro				
6	K	BGA	Oranienburg	6	K	BGA	Oranienburg
6	K	XLD	Oranienburg	6	K	XLD	Oranienburg
7	K	BGA	Ohio	7	K	BGA	Ohio
7	K	XLD	Ohio	7	K	XLD	Ohio
				8	H	XLD	Havana
9	K	BGA	Oranienburg & Braenderup	9	K	BGA	Oranienburg
9	K	XLD	Braenderup	9	K	XLD	Braenderup
10	H	BGA	Montevideo	10	H	BGA	Montevideo
10	H	XLD	Montevideo	10	H	XLD	Montevideo & Livingstone

*Media = Plating media used: BGA (Brilliant Green Agar); XLD (Xylose Lysine Deoxycholate).

partment of Agriculture (DOA) laboratory for testing. No eggs were damaged, cracked, or visibly soiled.

Of the 12 brands available for study, three were from US mainland producers and nine were locally produced. Fourteen dozen eggs were potentially available each week from each of the 12 brands, as two brands sold both white and brown eggs. Both white and brown eggs were sampled when available. Three brands on some weeks were not available at the designated supermarket locations. Unsuccessful attempts were made to locate these brands at other store locations. A total of 106 dozen eggs were sampled and cultured.

Isolation and identification of *Salmonella* from eggs were conducted using methodology from the US Food and Drug Administration,[9] with one variation. In addition to the recommended incubation temperature of 35 degrees Celsius (C) for samples in tetrathionate enrichment medium, a second incubation temperature (41 degrees C) was used.[10,11]

In order to avoid cross-contamination of samples, glassware was autoclaved prior to and between each use. Individual sterile pipettes and sterile gloves were used, and the pre-enrichment media were autoclaved.

The eggs from each dozen were separated into two flasks; one containing the shells and the other containing the magma (white and yolks). All samples were incubated for 18–24 hours in a lactose broth, pre-enrichment medium.[9]

Samples from each flask were then transferred to two flasks containing tetrathionate enrichment broth. One set of samples was incubated at 35 degrees C and the other at 41 degrees C.[10,11] After incubation for 24 hours, one loopful from each flask (magma and shell) was streaked onto plates of Xylose Lysine Desoxycholate Agar (XLD) and Brilliant Green Agar (BGA).[9] Isolates were serotyped using Centers for Disease Control methods.[12]

Results

Salmonellae were detected in 10 cartons (9.4 percent) of the 106 dozen eggs sampled. Positive samples were from shells only. *Salmonellae* were detected in samples from only three of the 12 brands examined. The brand with the highest weekly prevalence (6/8) was locally produced; the second highest (3/8) was a mainland brand; while the third highest (1/8) was a local brand.

Use of two enrichment incubation temperatures resulted in improved *Salmonellae* isolation rates. Three of the 10 dozen eggs testing positive came only from the enrichment medium incubated at 41 degrees C. One positive sample came only from the enrichment medium incubated at 35 degrees C. The remaining six dozen positive samples grew at both temperatures. Serotypes *braenderup, oranienburg, mbandaka, cerro, ohio, havana, montevideo,* and *livingstone* were detected (Table 1).

Discussion

An earlier egg survey done in Hawaii[13] as well as surveys conducted in New York,[14] Missouri,[15] British Columbia, [16] India,[17] and Saudi Arabia[18] demonstrated the prevalence of *Salmonella* on the surface of washed, commercially processed egg shells to be 0–2 percent. However, these surveys used only a single enrichment incubation temperature.

The use of two incubation temperatures for the enrichment medium increased the sensitivity for *Salmonella* detection in the present study. *Salmonella* would have gone undetected in three of the 10 dozen eggs had only the standard incubation temperature been used.

The US mainland brand eggs testing positive for *Salmonella* were traced to a USDA supervised plant in California. Upon inspection, no deficiencies were observed in this plant. The two brands of locally produced eggs were traced to one processor and a single farm. As a result of this study, the Hawaii State DOA inspected the processing plant. Equipment used in the egg washing and sanitizing process was found to malfunction. The company voluntarily agreed to cease operations until the deficiencies were corrected. The eggs were diverted to another facility for processing pending repair of the defective equipment. To date, the facility has not resumed operations as an egg processing plant. It is currently being used as a storage facility (personal communication DOA).

Recent reports of egg-associated salmonellosis have incriminated Grade A shell eggs that were considered to have been properly processed.[2–5,8] While transovarian transmission has also been implicated as a possible cause for the resurgence in egg-borne salmonellosis,[5,8] deficiencies in egg-processing procedures may be of more importance.

In Hawaii, the state DOA is the only state agency with regulatory authority over the egg industry. However, the scope of its activity is limited to the structural integrity of the eggs. Currently, there are no regulations that mandate active surveillance and monitoring of eggs for bacterial contamination. Our data indicate that visual inspection of eggs is not enough. Ongoing egg bacterial surveillance with improved laboratory methods, including the use of two enrichment incubation temperatures to increase *Salmonella* culture sensitivity, would allow for early detection of deficiencies in egg handling and processing, and could eliminate *Salmonella* egg contamination as a potential cause of disease in the community. ☐

Acknowledgments

Grateful acknowledgment is given to the following individuals for technical assistance and material support: James Foppoli, PhD, DVM, Veterinary Microbiologist, and Richard Chang, Microbiologist, Veterinary Laboratory Branch, Hawaii State Department of Agriculture; Arthur Oishi, Eggs and Feeds Program Specialist, and Samuel Camp, Manager, Commodities Branch, Hawaii State Department of Agriculture; and Velma Goo, MPH, Microbiologist, Laboratories Branch, Hawaii State Department of Health.

References

1. Cohen ML, Blake PA: Trends in foodborne salmonellosis outbreaks: 1963–1975. J Food Protect 1977; 40:798–800.
2. Update: Salmonella enteritidis infections in the Northeastern United States. MMWR 1987; 36:204–205.
3. Update: Salmonella enteritidis infections and grade A shell eggs—United States. MMWR 1988; 37:490,495,496.
4. Update: Salmonella enteritidis infections and grade A shell eggs—United States, 1989. MMWR 1990; 38:877–880.
5. St. Louis ME, Morse DL, Potter ME, *et al:* The emergence of grade A eggs as a major source of salmonella enteritidis infections. JAMA 1988; 259:2103–2107.
6. Pelzer KD: Salmonellosis. J Am Vet Med Assoc 1989; 195:456–463.
7. Williams JE, Dillard LH, Hall GO: The penetration patterns of *salmonella typhimurium* through the outer structure of chicken eggs. Avian Dis 1968; 12:445–466.
8. Sharp JCM: Salmonellosis and eggs. Br Med J 1988; 297:1557–1558.
9. Andrews WH, Poelma PL, Wilson CR: Isolation and identification of salmonella species. *In:* Bacteriological Analytical Manual of the Division of Microbiology, Center for Food Safety and Nutrition. US FDA, Sixth Edition. Arlington, Virginia: Association of Official Analytical Chemists, 1984: 7.01–7.18.
10. Bailey JS, Chiu JY, Cox NA, Johnston RW: Improved selective procedure for detection of salmonellae from poultry and sausage products. J Food Protect 1988; 51:391–396.
11. Van Schothorst M, Van Leusden FM: Comparison of several methods for the isolation of salmonellae from egg products. Can J Microbiol 1975; 21:1041–1045.
12. Rhoden DL: Isolation and identification of enterobacteriaceae in the clinical laboratory. Atlanta, GA: Centers for Disease Control, Bureau of Laboratories, Clinical Bacteriology Division, 1980.
13. Goo VYL, Ching GQL, Gooch JM: Comparison of brilliant green agar and Hektoen enteric agar media in the isolation of salmonellae from food products. Appl Microbiol 1973; 26:288–292.
14. Baker RC, Goff JP, Timoney JF: Prevalence of salmonellae on eggs from poultry farms in New York state. Poultry Sci 1980; 59:289–292.
15. Haque AKM, Vandepopuliere JM, Cotterill OJ: Microbial counts and salmonella in shell eggs from seven retail outlets. (abstract) Poultry Sci 1989; 68 (suppl 1):62.
16. March BE: Bacterial infection of washed and unwashed eggs with reference to salmonella. Appl Microbiol 1969; 17:98–101.
17. Panda PC: Salmonella contamination of shell eggs. Zentralbl Bakteriol [Naturwiss] 1975; 129:753–756.
18. Barbour EK, Nabbut NH: Isolation of salmonella and some other potential pathogens from two chicken breeding farms in Saudi Arabia. Avian Dis 1981; 26:234–244.

Agricultural Machine-Related Deaths

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ABSTRACT

Analysis of 1980-1985 death certificate data for the United States indicated that an average of 369 occupational deaths per year involved agricultural machinery as the external cause of death. Out of all agricultural machine-related deaths, tractors accounted for 69 percent. Over half of these tractor-related deaths were rollovers. There is a need for public health programs to affect greater use of rollover protective structures (ROPS) on farm tractors. (*Am J Public Health.* 1991;81:766–768)

Introduction

Fatalities among farm machine operators present a conspicuous injury target for public health action.[1–3] Farm tractors are known to be particularly deadly,[4–6] but prevention programs appear to be floundering.

As a first step toward the establishment of priorities for fatality prevention programs in agriculture, we decided to examine the National Traumatic Occupational Fatality (NTOF) data base maintained by the National Institute for Occupational Safety and Health (NIOSH).[7,8] Previous analysis of NTOF data for the years 1980-85 had shown that the Agriculture, Forestry, and Fishing industry had a fatality rate of 20.7 per 100,000 workers, a rate 2.6 times higher than the national average for all industries of 7.9 deaths per 100,000 workers.[7]

Methods

Death certificate data in NTOF were reviewed to identify persons killed while working with machines. Machine-related fatalities were identified by codes E919.0 to E919.9 according to ICD-9. Next, the type of machine involved in each machine-related fatality across all United States industries was ascertained by reviewing the description of the cause of death on the death certificate. All agricultural tractor-related fatalities were then categorized and compared by the Standard Industrial Classification (SIC) for the industry in which the incident occurred.[9] Age at death was determined for those fatalities associated with tractors, augers, hay balers, combines, and other agricultural machines.

TABLE 1—Machine-related Occupational Fatalities, All US Industries, 1980–85

Machine Type	Frequency*	Percent of Fatalities
Agricultural machines, E919.0	2216	43.8
Lifting machines, E919.2	1029	20.3
Mining, earth drilling machines, E919.1	467	9.2
Earthmoving, excavating machinery, E919.7	458	9.0
Other specified machinery, E919.8	441	8.7
Unspecified machinery, E919.9	237	4.7
Metalworking machinery, E919.3	88	1.7
Transmission machinery, E919.6	78	1.5
Woodworking machinery, E919.4	47	0.9
Total	5061	99.8**

*Values are based on the 90.6 percent of case records in the NTOF data file that have ICD codes.
**Sum differs from 100 due to rounding.

TABLE 2—Frequency of Agricultural Machine-Related Fatalities (E919.0), All US Industries, 1980–85

Agricultural Machine	Fatalities	Percent
Tractor	1523	69
Auger/elevator	61	3
Combine	53	2
Hay Baler	42	2
Brush hog/mower	40	2
Loader/skidder	37	2
Corn picker	26	1
27 other machines	108	5
Unspecified machine	326	15
Total	2216	101

*Sum differs from 100 due to rounding.

Results

The search of NTOF cases identified 5,061 machine-related fatalities coded E 919.0 to 919.9 (Table 1). Agricultural machines were involved in 2,216 deaths, an annual average of 369 deaths per year. The second ranking machine category on the list is lifting machines (cranes, forklifts, etc.) which averaged 170 fatalities annually, less than half that of agricultural machines.

The seven types of agricultural machines with greatest fatality frequency are listed individually in Table 2. Machines with a frequency less than 1 percent were combined into one category. The final category includes fatalities in which the type of machine was not specified on the death certificate. These data indicate that tractors are involved in the vast majority of agricultural machine-related fatalities. All other agricultural machines are involved in far fewer fatalities when compared to tractors.

Tractor overturns were the single most important event in fatalities involving tractor operators. Of the tractor-related fatalities in the NTOF data set, 52 percent were rollovers. Another 16 percent involved the victim being run over, such as instances in which an operator fell off the tractor and was run over. About 7 percent of the tractor-related fatalities were associated with the power-takeoff.

Farm tractors are used in many industries. For example, using farm tractors to pull cut timber in logging operations is considered to be a manufacturing industry activity. Of farm tractor-related fatalities, 59.3 percent occurred in the agriculture, forestry, and fishing industry, while manufacturing, services, and construction industries each contributed over 5 percent to the national death toll. Victims over 50 years old accounted for 62 percent of farm tractor-related fatalities, and victims over age 60 accounted for 44 percent of farm tractor-related fatalities.

Discussion

The findings reported here are similar to those reported in studies of farm fatalities in Wisconson[5] and Georgia.[6] All of these investigations found that tractor overturns accounted for the largest proportion of farm deaths (Wisconsin 52 percent; Georgia 76 percent; US 52 percent.)

Education, enforcement, and engineering control are often cited as the avenues for prevention of occupational injuries and fatalities. The mandatory education approach to farm tractor safety has not yet received sufficient emphasis. Some training is available through voluntary organizations such as the 4-H and Future Farmers of America. However such training is voluntary. It is unnecessary to pass a test, as is the case with obtaining a license to operate a motor vehicle, unless the tractor driver is age 14–16 and employed by someone not in the family.[10] For all other farm tractor drivers, no license of any kind is required. Furthermore there is no competency-based certification program for tractor operators.

The enforcement approach also has problems. In its annual appropriations bills for the Occupational Safety and Health Administration (OSHA) in the US Department of Labor, Congress has consistently attached a rider which denies OSHA the authority to inspect farms with fewer than 11 employees. This exempts 89 percent of US farms which may choose to comply or not comply with federal safety standards, free of an OSHA inspection. Unlike some consumer products, which may not be sold in the US if they do not meet regulations of the Consumer Product Safety Commission, the sale of farm machinery is not regulated for safety. Because most farm workers are not covered by OSHA enforcement, regulating farm machine safety at the point of sale has been suggested as a promising way to protect farm workers from machine hazards.

The engineering approach involves efforts at preventing rollovers, as well as protecting operators if a rollover does occur by fitting tractors with rollover protective structures (ROPS). Much effort has already gone into the design of ROPS for tractors, and industry-accepted voluntary standards call for the provision of ROPS on new tractors. OSHA has a requirement that employers must have ROPS on new tractors built since October 25, 1976. One of the difficulties encountered in this approach is the fact that farm tractors tend to be used for so many years that replacement of older tractors with newer, safer tractors is proceeding very slowly. There is also a problem with some farmers removing ROPS from new tractors. Considering all these factors, during the next 10 or 20 years it is unlikely that the percentage of tractors with ROPS will increase rapidly unless

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some significant changes take place. Although engineering has provided the technology for preventing most fatalities due to tractor rollovers, it appears that voluntary implementation is very slow.

In Sweden a very significant reduction in tractor rollover fatalities has been brought about by requiring rollover protection on farm tractors.[11] Between 1961 and 1983, Sweden was able to drop its annual fatality rate for tractor overturning from 12 per 100,000 farmers to 1 per 100,000 farmers. This was accomplished through regulations introduced periodically between 1959 and 1981.

It will take cooperative action, including input from farmers, to develop creative prevention programs to solve this difficult problem. Education, enforcement, and engineering will work if they are supported by the common interest of a united agricultural public health community. Achieving this unity is a crucial goal for the 1990s. □

References

1. Baker S, O'Neill B, Karpf R: The Injury Fact Book. Lexington, MA: Lexington Books, 1984.
2. Purschwitz M, Field W: Scope and magnitude of injuries in the agricultural workplace. *In*: Proc Winter Meeting of the American Society of Agricultural Engineers, Chicago, IL, 1987; Paper no. 87-5514. Available from American Society of Agricultural Engineers.
3. National Academy of Sciences, Committee on Trauma Research: Injury in America: A Continuing Public Health Problem. Washington, DC: National Academy Press, 1985.
4. National Safety Council: Accident Facts, 1988 Ed. Chicago, IL: National Safety Council, 1988.
5. Karlson T, Noren J: Farm tractor fatalities: The failure of voluntary safety standards. Am J Public Health 1979; 69:146–149.
6. Goodman RA, Smith JD, Sikes RK, Rogers DL, Mickey JL: Fatalities associated with farm tractor injuries: An epidemiologic study. Public Health Rep 1985; 10:329–333.
7. National Traumatic Occupational Fatalities: 1980–85. Morgantown, WV: Division of Safety Research, National Institute for Occupational Safety and Health: 1989.
8. Bell CA, Stout NA, Bender TR, Conroy CS, Crouse WE, Myers JR: Fatal occupational injuries in the United States, 1980–1985. JAMA 1990; 263:3047–3050.
9. Office of Management and Budget: Standard Industrial Classification Manual. Washington, DC: Govt Printing Office, 1972.
10. US Department of Labor: Occupations in agriculture particularly hazardous for the employment of children below the age of 16. 29 CFR 570.70-.71, Washington, DC: Govt Printing Office, 1988.
11. Springfeldt B, Thorsen J: Mitigation of personal injuries caused by overturning of farming tractors. *In*: Proceedings of the 31st Annual Meeting of the American Association for Automotive Medicine, New Orleans, September 28–30, 1987; 229–236. Available from American Association for Automotive Medicine.

A Cross-sectional Study of Pulmonary Function in Autobody Repair Workers

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ABSTRACT

This study evaluated pulmonary function in workers from 39 autobody repair shops. Based on 152 White male workers with known smoking status, the mean percent predicted FEV_1, FVC, and FEV_1/FVC were 93.6, 96.8, and 96.6, respectively. Twenty-three percent of workers had a FEV_1/FVC ratio less than the fifth percentile. Isocyanate levels ranged from nondetectable to 0.06 parts per million (STEL = 0.02 ppm). No shop had an adequate respiratory protection program. We concluded that there was an increase in abnormal pulmonary function in autobody workers. Three recommendations were made to shop owners: functional paint booths should be maintained, respiratory protection programs should be developed, and isocyanate-free paints should be used. (*Am J Public Health.* 1991; 81:768–771)

Introduction

Approximately 700 autobody repair shops employ an estimated 3,500 individuals in Minnesota (Minnesota Department of Labor Relations, Personal Communication). Autobody repair involves hammering dents, removing damaged parts, welding, grinding, abrasive blasting, filling with styrene putty, sanding, and spray painting.[1,2] Workers may be exposed to hazards including isocyanates, metals in paint, solvents, dusts, and noise.[2,3] Despite possible adverse health effects,[4–7] there has been little epidemiologic investigation of the autobody repair industry.[1,8–10] This study examines pulmonary function in autobody repair workers.

Methods

Sampling Protocol

Autobody repair shops listed in the Twin Cities Yellow Pages were randomly selected for study. Shops were included in the study if: 1) they had 20 or fewer employees; 2) at least half of establishment income was from autobody repair; and 3) they were located within the Twin Cities metropolitan area. Shop owners were asked questions regarding the inclusion criteria. The owners of eligible shops were asked to participate.

A questionnaire was given to each shop manager regarding shop age, size, number of cars repaired, ventilation, and repair materials. Sampling for toluene diisocyanate (TDI), total particulates, and solvents (acetone, benzene, methyl ethyl ketone, toluene, xylene, trichloroethylene, and mineral spirits) was done in all

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TABLE 1—Characteristics of 39 Autobody Repair Shops

	Mean	Standard Deviation	Range
Number of Workers	3.9	2.6	1–12
Years in Business	18.8	16.0	<1–70
Age of Building	26.4	21.9	<1–90
Repaired each Month	55.4	45.2	9–200

shops. Total isocyanates [TDI and methylene isocyanate (MDI)] were evaluated in 16 shops, lead levels in nine shops, and silica levels in seven shops. Sampling was done in a central work area for three to six hours. Pumps were calibrated before and after each sampling period. Analyses were done according to the National Institute for Occupational Safety and Health (NIOSH) Manual of Analytic Methods.[11]

A self-administered respiratory and work history questionnaire was given to each worker and reviewed with them for completeness. Using American Thoracic Society[14] criteria, pulmonary function was evaluated at the start and end of the work day with a Collins water-sealed spirometer. Measurements consisted of the forced expiratory volume at one second (FEV_1), forced vital capacity (FVC), and the FEV_1/FVC ratio. Spirometric values were adjusted for ambient temperature, barometric pressure, age, sex, and height.

Data Analysis

The shops and workers were characterized using descriptive statistics. Mantel-Haenszel odds ratios, stratified by smoking status (current, past, never) were computed for categorical variables and combined into a single summary odds ratio. Bartholomew's test for trend was used to evaluate the effect of work practices on the proportion of workers with cough or wheeze. Each of the independent variables (e.g. respirator use while sanding) was ordered as: never, sometimes, always.[15]

Percent of predicted FEV_1, FVC, and FEV_1/FVC were calculated for White males using the equations of Crapo, *et al.*[16] An "abnormal" pulmonary function test was one below the fifth percentile for age, height, and sex. The fifth percentile is a value that is approximately 80 percent of predicted for the FEV_1 and FVC, and approximately 90 percent of predicted for the FEV_1/FEC.[17] A one sample binomial test was used to evaluate the prevalence of abnormal pulmonary function tests. Morning and afternoon pulmonary function were compared using paired t-tests. Predictors of pulmonary function were evaluated using stepwise linear regression.[18]

Results

Shop Characteristics

Of the 128 shops contacted, seven (5 percent) were out of business, and 52 (41 percent) were not eligible. Of the 69 remaining shops, 59 were asked to participate of which 39 (66 percent) agreed. Ten shops were not asked to participate because *a priori* study enrollment goals had been met.

Table 1 shows the characteristics of the 39 shops studied. Two shops were one-person operations, and all but one were independently owned and operated. Even though most shops (72 percent) did sandblasting, this was rarely done for more than one hour per week. Ten shops had paint booths in a poor or nonworking condition.

Ambient levels for silica were below the detection limit for the seven shops tested. All solvent levels were below the current American Conference of Governmental Industrial Hygienists (ACGIH) guidelines. Total particulates ranged from 0.4 mg/m^3 to 14 mg/m^3. Particulate levels exceeded 5 mg/m^3 in four shops, and 10 mg/m^3 in one; the current ACGIH guideline for nuisance dusts.[19] Proton-induced x-ray analysis of ambient dusts revealed the presence of small quantities of 20 metals. No ambient lead was detected in the nine shops that were evaluated.

Isocyanate samples were collected from 32 shops. Two samples were lost due to laboratory error. Eight-hour, time-weighted average total isocyanate levels ranged from not detected to 0.06 ppm (mean = 0.005 ± 0.008). The current ACGIH recommended level for TDI and MDI is 0.005 ppm.[20] The highest and lowest isocyanate levels were from the same shop on different days. In three shops, total isocyanate levels were slightly above current ACGIH guidelines. Two to twelve times the current guideline was exceeded in another four shops.

Of 164 workers, 162 (99 percent) completed questionnaires and 159 (97 percent) performed acceptable morning and 149 (91 percent) acceptable afternoon pulmonary function tests. The mean age was 31.6 years (±11.3), 157 (97 percent) were White and 160 (99 percent) males. The mean years worked in the autobody industry was 11.4 (±9.7). Cigarette smoking was common: 67 (41 percent) current, 30 (18 percent) former, 63 (39 percent) never smokers, and two (1 percent) unknown.

Of the 122 workers who spray painted at least one hour per week, 4 percent never used a respirator while painting, 33 percent sometimes did, and 63 percent always used one. For the 96 workers who did sandblasting these percentages of respirator use were 25 percent, 33 percent, and 42 percent, respectively.

Table 2 shows the number of individuals whose pulmonary function was below the fifth percentile and the mean values of the pre-work percent predicted FEV_1, FVC, and FEV_1/FVC stratified by smoking status. Among 60 workers who had never smoked, five (8 percent) had baseline FEV_1 or FVC measurements below the fifth percentile. Fourteen (23 percent) had an FEV_1/FVC below the fifth percentile ($p < 0.001$). No significant change in pulmonary function was seen between the morning and afternoon shifts. In addition, no relationship was seen between shop isocyanate levels and pulmonary function.

By regression analysis, a nonfunctioning paint booth ($\hat{B} = -8.35$), smoking ($\hat{B} = -3.84$), years worked in the autobody industry ($\hat{B} = -0.20$), hours per week sanding ($\hat{B} = 0.21$), doing paperwork ($\hat{B} = 0.20$), and assembly ($\hat{B} = 0.18$) significantly predicted the percent predicted FEV_1/FVC ($R^2 = 0.19$). Decrements in both the percent predicted FEV_1 ($R^2 = 0.20$) and FVC ($R^2 = 0.11$) were significantly associated with years worked in the autobody industry and a nonfunctioning paint booth.

Summary odds ratios showed a positive, non-significant association between the presence of cough and wheezing* and 1) failure to have a functioning paint booth, 2) failure to use a respirator, and 3) spray painting and/or sandblasting. Using

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*Cough was defined as coughing four to six times per day at least four days per week. Wheezing was defined as wheezing on most days or nights.

TABLE 2—Percent Predicted Morning Pulmonary Function and Number of Workers Whose Pulmonary Function Was Less Than the Fifth Percentile by Smoking Status*

Smoking Category (N)	Mean	Standard Deviation	Number with Abnormal Pulmonary Function (%)**
Never Smoker (N = 60)			
FEV_1	93.6	11.4	5 (8)
FVC	96.8	11.4	5 (8)
FEV_1/FVC	96.6	7.0	14 (23)
Former Smoker (N = 30)			
FEV_1	91.6	13.8	6 (20)
FVC	95.6	13.1	3 (10)
FEV_1/FVC	95.8	7.4	8 (27)
Current Smoker (N = 62)			
FEV_1	90.0	15.3	11 (18)
FVC	96.3	13.6	4 (6)
FEV_1/FVC	93.1	10.0	18 (29)

*A total of 159 workers completed at least one acceptable spirometric examination. Of these, five non-White workers and two workers whose smoking status was unknown were excluded from the analysis.

**Abnormal is defined as corresponding to less than the fifth percentile. For the FEV_1 and FVC this is approximately 80 percent of predicted and for the FEV_1/FVC ratio, approximately 90 percent of predicted.

a chi square test for trend, for nonsmokers, the presence of wheezing significantly ($p < 0.05$) increased across the categories of respirator use (always, sometimes, never) while spray painting and for coughing and wheezing while sandblasting. No significant trends were seen for respiratory symptoms and respirator use while sanding.

Discussion

Two-thirds of all shops contacted agreed to participate. Although no information was available on shops that refused to participate, it is unlikely that they were cleaner than the ones tested. Shops were generally dusty and strong solvent odors were common. Improper respiratory protection was often used, when used at all. These findings are similar to those of Tuskes and Key[1] and Rose, *et al.*[8]

Isocyanate levels were consistent with the mild, but significant, decline in pulmonary function and the presence of respiratory symptoms seen.[20,28] Years working in the autobody industry, and working in a shop with a nonfunctioning paint booth, along with smoking, were associated with a decrement in the FEV_1/FVC indicative of obstructive disease.[30,31] Time spent doing tasks not involving painting or priming (i.e. sanding, doing paperwork, and assembly) were positively associated with the FEV_1/FVC. No relationship was found between pulmonary function and ambient isocyanate levels. This was not surprising given the day-to-day variability in the type and quantity of work done in each shop. The failure to find acute pulmonary changes may be due to the migration of sensitized workers who may have had to find other employment or the fact that it was unusual to visit a shop on a Monday. We did not monitor over the course of the week for delayed asthmatic reactions.[26]

The environment in autobody repair shops may be improved in several ways. Shops should maintain functional paint booths for spray painting, and the spray painting of vehicles in open shop areas should be avoided. Shops should have complete respiratory protection programs and use less hazardous, isocyanate-free paints. ☐

Acknowledgments

This study was made possible by a grant from the Park-Nicollet Medical Foundation, St. Louis Park, Minnesota. Special assistance and equipment were provided by the Division of Business and Occupational Health, Park-Nicollet Medical Center. The authors thank Dr. Jack Brondum at the Minnesota Department of Health and Dr. Ian Greaves and Mary Dolan at the University of Minnesota for their comments and assistance.

References

1. Tuskes PM, Key MM: Potential hazards in small business: A gap in OSHA protection. Appl Ind Hyg 1988; 3:55–57.
2. Karches GJ: Health and safety guide for auto repair and body shops. Washington, DC: US Department of Health, Education, and Welfare, February 1975.
3. National Institute for Occupational Safety and Health: Safety and health in autobody repair shops. Cincinnati: US Department of Health and Human Services, 1977 (NIOSH Pub. No. 77-2290).
4. Rye WA: Human responses to isocyanate exposure. JOM 1973; 15:306–307.
5. Banks DE, Butcher BT, Salvaggio JE: Isocyanate-induced respiratory disease. Ann Allergy 1986; 57:389–396.
6. World Health Organization: Environmental Health Criteria 75: Toluene Diisocyanates. Geneva: WHO, 1987.
7. World Health Organization: Environmental Health Criteria 3: Lead. Geneva: WHO, 1977.
8. Rose DN, Cummings DE, Molinaro J, *et al:* Screening for lead toxicity among autobody repair workers. Am J Ind Med 1982; 3:405–412.
9. Elofson S, Gamberle F, Hindmarsh T, *et al:* Exposure to organic solvents: A cross sectional investigation of occupationally exposed car and industrial spray painters with special reference to the nervous system. Scand J Work Environ Health 1980; 6:239–273.
10. Husman K: Symptoms of car painters with long-term exposure to a mixture of organic solvents. Scand J Work Environ Health 1980; 6:19–32.
11. National Institute for Occupational Safety and Health: NIOSH Manual of Analytic Methods, 3rd Ed. Cinncinnati: US Department of Health and Human Services, 1984 (Pub. No. 84-100).
12. Ferris BG: Epidemiology Standardization Project II: Recommended respiratory disease questionnaires for use with adults and children in epidemiologic research. Am Rev Respir Dis 1978; 118:7–53.
13. Blumberg WE, Eidsenger J, Lamola A, *et al:* The hematoflurometer. Clin Chem 1977; 23:270–274.
14. Ferris BG: Epidemiology Standardization Project III: Recommended standardized procedures for pulmonary function testing. Am Rev Respir Dis 1978; 118:55–88.
15. Fleiss JL: Statistical Methods for Rates and Proportions, 2nd Ed. New York: John Wiley and Sons, 1981.
16. Crapo RO, Morris AH, Gardner RM: Reference spirometry values using techniques and equipment that meets ATS recommendations. Am Rev Respir Dis 1981; 123:659–664.
17. Pennock BE, Cottrell JJ, Rogers RM: Pulmonary function testing: What is "normal?" Arch Intern Med 1983; 143:2123–2127.
18. SPSS Inc: SPSS-X User's Guide. Chicago, IL: SPSS Inc, 1988; 849–890.
19. American Conference of Governmental and Industrial Hygienists: Threshold limit values and biological exposure indices for 1987 and 1988. Cincinnati: ACGIH, 1987.
20. Bernstein IL: Isocyanate induced pulmonary disease. J Allergy Clin Immunol 1982; 70:24–31.
21. Zammitt-Tabona M, Sharkin M, Kijek K, Chan H, Chan-Yeung M: Asthma caused by diphenylmethane diisocyanate in foundry workers. Clinical, bronchial, provocation and immunologic studies. Am Rev Respir Dis 1983; 128:226–230.
22. Banks DE, Bankman HW, Butcher BT, Hammad YY, Rando RJ, Glindmeyer HW,

Jones RN: Absence of hyperresponsiveness to methacholine in a worker with methylene diphenyl diisocyanate (MDI)-induced asthma. Chest 1986; 3:389–393.

23. Baur M, Dewair M, Rommelt H: Acute airway obstruction followed by hypersensitivity pneumonitis in an isocyanate (MDI) exposed worker. JOM 1984; 26:285–287.
24. Peters JM, Murphy RLH, Ferris BG: Ventilatory function in workers exposed to low levels of toluene diisocyanate. Br J Med 1969; 26:115–120.
25. Peters JM: Studies of isocyanate toxicity: Cumulative pulmonary effects in workers exposed to toluene diisocyanate. Proc R Soc Med 1970; 63:372–375.
26. Peters JM, Murphy LHM, Pagnatto LD, *et al:* Respiratory impairment in workers exposed to safe levels of toulene diisocyanate. Arch Environ Health 1970; 20:364–367.
27. Wegman DH, Peters JM, Pagnatto LD, *et al:* Chronic pulmonary function loss from exposure to toluene diisocyanate. Br J Ind Med 1977; 34:196–200.
28. Diem JE, Jones RN, Hendrick DJ, *et al:* Five-year longitudinal study of workers employed in a new toluene diisocyanate manufacturing plant. Am Rev Respir Dis 1982; 126:420–428.
29. Musk WJ, Peters JM, Di Bernardinis L, Raymond RLH: Absence of respiratory effects in subjects exposed to low concentrations of TDI and MDI. JOM 1982; 24:746–750.
30. Gee JB, Morgan WK: A 10-year follow-up study of a group of workers exposed to isocyanates. JOM 1985; 34:263–271.
31. Higgins MW, Keller JB: Seven measures of ventilatory lung function: Population values and a comparison of their ability to discriminate between persons with and without chronic respiratory symptoms and disease. Am Rev Respir Dis 1973; 108:258–272.

Measuring Physical Activity With a Single Question

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ABSTRACT

Using 1,004 subjects enrolled in a worksite health promotion program, this report evaluated the validity of a single question about participation in regular exercise. Measured at baseline, this one question had a significant age-adjusted association with body mass index ($p < 0.0001$ in women and $p = 0.001$ in men), HDL cholesterol ($p < 0.0001$ in women), and oxygen capacity ($p = 0.0007$ in women and $p = 0.002$ in men). Thus, one self-reported question can provide useful information about who is and who is not participating in regular exercise. The potential validity of a single exercise question is particularly relevant in complex epidemiologic studies where lengthy questionnaires highlight the importance of brief instruments. (*Am J Public Health.* 1991;81:771–773)

Introduction

Through its association with low levels of HDL (high-density lipoprotein) cholesterol, obesity, and other physiologic parameters, physical inactivity has been linked to increased morbidity and mortality from a variety of diseases. These include cardiovascular diseases,[1] diabetes mellitus,[2] osteoporosis,[3] and even breast cancer[4] and colon cancer[5]. In light of these etiologic associations, the measurement of physical activity has become a standard component of epidemiologic research, with at least 30 different measurement techniques having been described.[6] Unfortunately, many of these are complex instruments that are associated with excessive costs and time commitments that make them impractical in large-scale epidemiologic studies.[6,7] Thus, there is great value in establishing the validity of brief, easily obtained measures of physical activity.

It is the purpose of the present report to discuss the utility of a single exercise question that was generated by the St. Louis Working Hearts Program. That question was "Do you currently participate in any regular activity or program (either on your own or in a formal class) designed to improve or maintain your physical fitness?" We will investigate the validity of this question by evaluating its association with three parameters that are known to be associated with physical activity: body mass index (BMI), HDL cholesterol, and oxygen capacity.

Methods

The data discussed herein were generated by Working Hearts, a four-year collaboration between Washington University in St. Louis and a large midwestern communications corporation. All employees at 17 divisions of the corporation were eligible to participate in a multifaceted two-year cardiovascular risk reduction intervention. The intervention included individualized counseling, worksite promotional activities, and 25 awareness seminars and workshops that focused on weight loss, cholesterol reduction, hypertension, exercise, stress reduction, diet, and smoking cessation. Of particular relevance is the fact that the exercise component of the intervention included discussions and workshops, but no regular exercise program. Data on three of the four variables pertinent to this report—exercise behavior, BMI, and HDL cholesterol levels—were sought from all study participants. BMI was de-

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TABLE 1—Oxygen Capacity and Baseline Body Mass Index and HDL Cholesterol by Exercise Status at Baseline (Data are expressed as mean ± standard deviation)

	Exercise Status		
Panel A: Women	No (N = 517)	Yes (N = 154)	Age-Adjusted p-value
Oxygen Capacity[1]	28.8 ± 7.5	32.3 ± 7.2	0.0007
Body Mass Index	26.7 ± 6.1	24.2 ± 4.4	0.0001
HDL Cholesterol	57.0 ± 13.4	61.5 ± 14.3	0.0001
Panel B: Men	No (N = 201)	Yes (N = 114)	Age-Adjusted p-value
Oxygen Capacity[1]	33.8 ± 7.5	40.3 ± 8.3	0.002
Body Mass Index	27.6 ± 4.0	26.1 ± 3.2	0.001
HDL Cholesterol	47.2 ± 12.2	48.2 ± 10.2	0.455

[1] Oxygen capacity data were available on 81 women and 33 men involved in vigorous exercise and 155 women and 35 men not involved in vigorous exercise. The unit of measurements is ml/kg/min.

fined as weight (in kg) divided by height (in meters) squared. Oxygen capacity, in ml/kg/min, was measured on a self-selected subset of 304 subjects using a sub-maximal bicycle test. Results, expressed as mean ± standard deviation, are analyzed separately for males and females. Analysis of covariance determined whether there were significant age-adjusted differences in the three relevant parameters between exercising and non-exercising subjects. To analyze changes over time, the 591 participants who provided both baseline and final exercise data were divided into four groups as follows: Group NN responded "no" at both times (N = 281, 47.5 percent); Group YY responded "yes" at both times (N = 130, 22.0 percent); Group NY reported initiating exercise (N = 118, 20.0 percent); and Group YN reported cessation of regular exercise (N = 62, 10.5 percent).

Results

The Working Hearts data set contained 1,004 subjects whose age was 36.6 ± 9.5 years; 73.1 percent were female. Among the 986 subjects who provided baseline exercise information, 32 percent (N = 316) reported participation in regular exercise. Subjects who provided oxygen capacity data (N = 304) were younger (p = 0.001 in women, p = 0.012 in men), had a lower baseline BMI (p = 0.036 in women, p = 0.049 in men), and were more likely to exercise at baseline among women (p = 0.008) but not among men (p = 0.265), than those not providing oxygen capacity data. However, after age adjustment, logistic regression found that BMI had no independent association with the availability of bicycle test data in either sex, but that baseline exercise was still significant in women (p = 0.030). The availability of bicycle test data was not associated with baseline HDL cholesterol in either sex.

Table 1 provides the key data summarizing the validity of the simplified exercise measure we have utilized. It indicates that at baseline, and after age adjustment, subjects who said they were exercisers had a lower BMI (p < 0.0001 in women and p = 0.001 in men), greater oxygen capacity (p = 0.0007 in women and p = 0.002 in men), and had a higher HDL cholesterol (p < 0.0001 in women) than those who did not report participation in regular exercise.

Using the four groups defined in the Methods section, Table 2 contains the mean change from baseline to final measurement in both BMI and HDL cholesterol. Oxygen capacity was not evaluated in this context because it was measured at only one time point. Following age adjustment, mean HDL cholesterol was not the same in all four groups of men (p = 0.007). Statistical contrasts show that this reflected an *increase* in HDL in group YY (2.2 ± 11.5, N = 58) that was significantly

TABLE 2—Changes in Body Mass Index and HDL Cholesterol by Exercise Status[a] (Data are expressed as mean ± standard deviation)

Panel A: Women	Group NN (N = 209)	Group NY (N = 92)	Group YN (N = 43)	Group YY (N = 69)	Age-Adjusted p-value[b]
Change in body Mass Index	0.59 ± 1.9	0.05 ± 2.2	0.69 ± 2.2	0.23 ± 1.7	0.100
	Contrasts:[c] Group NN vs Group NY: p = 0.028				
Change in HDL Cholesterol	−1.3 ± 10.7	−2.2 ± 6.7	−3.3 ± 11.5	−2.8 ± 7.9	0.655
Panel B: Men	**Group NN (N = 72)**	**Group NY (N = 26)**	**Group YN (N = 19)**	**Group YY (N = 61)**	**Age-Adjusted p-value[b]**
Change in body Mass Index	0.33 ± 1.3	−0.47 ± 1.5	−0.02 ± 0.87	−0.08 ± 2.5	0.218
Change in HDL Cholesterol	−3.0 ± 6.4	−1.7 ± 6.4	−1.0 ± 4.9	2.2 ± 11.5	0.007
	Contrasts:[c] Group NN vs Group YY: p = 0.0006				

[a] Exercise groups are: Group NN = No exercise at baseline or final screening; Group NY = Not exercising at baseline, exercising at final screening; Group YN = Exercising at baseline but not at final screening; Group YY = Exercising at both time points.

[b] Based on analysis of variance testing equality in all four groups and adjusting for age.

[c] Not adjusted for multiple comparisons.

different from the *decrease* in HDL cholesterol in group NN (−3.0 ± 6.4, p = 0.0006, N = 70). Although the overall age-adjusted ANOVA showed no difference in BMI among the four groups of women (p = 0.100), it is worth noting that the statistical contrast comparing the increase in BMI in group NN (0.59 ± 1.9) with the small change in group NY (0.05 ± 2.2) was significant (p = 0.028).

Discussion

These results indicate that among Working Hearts subjects, a single self-reported measure of participation in regular exercise at baseline was significantly associated, after age adjustment, with body mass index and oxygen capacity in both sexes and with HDL cholesterol in women. Thus, our data support several other studies[8-10] which have suggested that simple self-reported instruments can yield valid information about participation in regular exercise. Based on only two exercise questions, for example, the Lipid Research Clinics Study[8] found that subjects who reported regular vigorous exercise had higher HDL cholesterol, lower resting heart rates, and longer exercise test durations than those not reporting regular exercise. Siconolfi et al[9] found that a single question concerning the self-reported frequency of exercise that induced sweating was associated with maximal oxygen uptake.

If a prime focus of a study is to gain detailed and precise information about levels of physical activity, extensive exercise questionnaires are required. Our results suggest, despite some positive associations in Table 2, that simplified exercise measures may also be inadequate when changes over time are of primary interest. However, as with the reports noted above,[8-10] our data argue strongly that one or two self-administered questions can provide a valid measure of who is and who is not currently involved in regular exercise. Thus, when extensive questionnaires place great demands on participants in a multifaceted epidemiologic study, and when detailed information about exercise behavior is not essential, a few simple exercise questions may be an appropriate alternative to the complex instruments that are frequently utilized. ☐

References

1. Powell KE, Thompson PD, Caspersen CJ, *et al*: Physical activity and the incidence of coronary heart disease. Annu Rev Public Health 1987; 8:253–287.
2. Vranic M, Berger M: Exercise and diabetes mellitus. Diabetes 1979; 28:147–163.
3. Montoye HJ, McCabe JF, Metzner HL, *et al*: Physical activity and bone density. Human Biol 1976; 48:599–610.
4. Frisch RE, Wyshak G, Albright NL, *et al*: Lower prevalence of breast cancer and cancer of the reproductive system among former college athletes compared to non-athletes. Br J Cancer 1985; 52:885–891.
5. Gehardsson M, Norell SE, Kiviranta H, *et al*: Sedentary jobs and colon cancer. Am J Epidemiol 1986; 123:775–780.
6. Laporte RE, Montoye HJ, Caspersen CJ: Assessment of physical activity in epidemiologic research: Problems and prospects. Public Health Rep 1985; 100:131–146.
7. Montoye HJ, Taylor HL: Measurement of physical activity in population studies: A review. Human Biol 1984; 56:195–216.
8. Haskell WL, Taylor HL, Wood PD, Schrott H, Heiss G: Strenuous physical acitvity, treadmill exercise test performance and plasma high-density lipoprotein cholesterol: The Lipid Research Clinics Program Prevalence Study. Circulation (suppl IV) 1980; 62:IV53–IV61.
9. Siconolfi SF, Lasater TM, Snow RC, Carleton RA: Self-reported physical activity compared with maximal oxygen uptake. Am J Epidemiol 1985; 122:101–105.
10. Godin G, Jobin J, Bouillon J: Assessment of leisure time exercise behavior by self-report: A concurrent validity study. Can J Public Health 1986; 77:359–362.

Public Health and the Law

OSHA's Four Inconsistent Carcinogen Policies

Background

No issue has proven more contentious in occupational health policy than the control of chemical carcinogens. The Occupational Safety and Health Administration (OSHA) has focused much of its efforts on cancer risks, both through substance-specific regulations and through broader efforts to establish generic policies and work practices. The 1980 Generic Carcinogen Policy was conceptualized as the centerpiece of OSHA's shift from substance-specific to generic regulations covering hundreds if not thousands of substances. Despite adverse judicial rulings and a generally anti-regulatory political climate, OSHA continued to pursue a generic approach to occupational health hazards during the 1980s, particularly through its 1983 Hazard Communication Standard and 1989 Air Contaminants Standard. The legal and political wrangling have left deep scars on OSHA's strategy, however. While consistency and comprehensiveness were once the principles underlying OSHA's efforts to control carcinogens, the current collection of policies and regulations is remarkable for the inconsistent and incomplete way in which suspect chemicals are treated.

In this paper, we analyze OSHA's four carcinogen strategies, as embodied in the Generic Carcinogen Policy, the substance-specific carcinogen regulations, the Hazard Communication Standard, and the Air Contaminants Standard. Two issues are of particular interest.

- Given the inherent methodological limitations to epidemiological data on occupational and environmental cancer, a major scientific and policy debate has developed over the appropriate use in human risk assessment of laboratory evidence on chemical carcinogenesis in animals. The four OSHA policies exhibit quite different positions on this debate.
- The choice between direct regulation and indirect labor market pressure, and the stringency of those exposure limits that are imposed, reflect different attitudes toward the appropriate balance of economic costs and health benefits for occupational health regulations. OSHA's four policies reflect dramatically different balancing efforts.

We begin with the Generic Carcinogen Policy, which remains OSHA's formal policy on the issue but which has been blocked by administrative stays and never incorporated into risk management programs. The substance-specific cancer standards are then analyzed in terms of their comprehensive treatment of a few public health hazards and neglect of many others. While partially an attempt to preempt stronger state and local regulations, the Hazard Communication Standard is in many ways both comprehensive and innovative, covering a wide range of chemicals and imposing duties on employers that could potentially lead to substitution of alternative products and processes. Given its current prominence as the centerpiece of OSHA's regulatory program, the Air Contaminants Standard receives the greatest emphasis. Containing its own internal inconsistencies, the Air Contaminants Standard embodies the best and the worst of OSHA's new regulatory philosophy and its strategy for controlling chemical carcinogens in the workplace.

The Generic Carcinogen Policy

OSHA's Generic Carcinogen Policy[1] was a response to two complementary concerns that emerged over the course of the 1970s and came to dominate the agency's agenda. OSHA had devoted a substantial portion of its resources to the regulation of individual carcinogens but had achieved only meagre results. The agency leadership faced growing demand for an accelerated regulatory timetable from Congress, organized labor, and environmental groups. Of equal significance, perhaps, was the competition among several federal agencies for leadership in formulating governmental policies with respect to chemical carcinogens. Proposed in 1977 and promulgated in 1980, OSHA's Generic Carcinogen Policy embodied a highly protective approach to chemical carcinogenesis. Based on the most conservative set of assumptions concerning methods of cancer induction and the most stringent set of requirements for exposure controls, the 1980 policy represents the high water mark of governmental enthusiasm for regulating occupational and environmental carcinogens.

OSHA devoted much of its energies during the 1970s to developing standards regulating exposure to occupational carcinogens, including asbestos[2] in 1972, vinyl chloride[3] and a group of 14 chemicals[4] in 1974, coke oven emissions[5] in 1976, plus benzene,[6] dibromochloropropane (DBCP),[7] inorganic arsenic,[8] and acry-

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lonitrile[9] in 1978. All of these except the DBCP and acrylonitrile standards underwent court challenges from the regulated industries, with lengthy delays and threats of reversal. A number of "science policy" issues reappeared at each challenge, including the relevance of animal data for human carcinogenesis, the importance of negative study results compared to positive results, the correlation between induction of benign and malignant tumors, and the purported existence of a threshold for carcinogenic effects. These same policy issues were being raised at EPA with respect to pesticides covered by the Federal Insecticide, Fungicide, and Rodenticide Act[10] and toxic air pollutants covered by the Clean Air Act.[11] After some discussions with EPA over the possibility of a joint carcinogen policy, OSHA decided to proceed with its own standard. The history of the OSHA policy and its relationship with initiatives at other regulatory agencies has been described in detail in several publications.[12–17]

The Generic Carcinogen Policy established a categorization system for known or suspected chemical carcinogens, with immediate regulatory action being proposed for substances falling into Category I. This category would be composed of substances that increased the incidence of benign or malignant tumors or decreased the latency period between exposure and onset in humans, in two animal species, in a single mammalian species in two or more independent experiments, or in a single experiment if supported by evidence from short-term tests. Within the universe of substances falling into Category I, the policy established a priority setting mechanism whereby the agency would select 10 substances for comprehensive standard setting at any one time, to include requirements for exposure limits, monitoring, worker training, and other factors. In 1980, OSHA issued a candidate list of 204 chemicals for review as possible candidates under the terms of the policy. The exposure limits were to be set at the lowest "feasible" level, which was interpreted by the agency as accepting any level of compliance costs short of causing massive economic dislocation and job loss in the regulated industry.

The weakening of the Generic Carcinogen Policy began almost as soon as it was promulgated. In July of 1980, the United States Supreme Court upheld the decision by the Fifth Circuit Court of Appeals to vacate the standard governing occupational exposure to benzene, on the grounds that OSHA had not proven that exposures to benzene at the level regulated by the standard posed a "significant risk" to employees.[18] This ruling implied that any standard not demonstrating significant risk, presumably through the emerging quantitative risk assessment methodologies, would be overturned. The types of evidence sufficient to qualify chemicals for OSHA's Category I did not demonstrate "significant risk" without further analysis. In January of 1981, OSHA issued a revised Generic Carcinogen Policy that mandated a two-part approach to characterization of individual carcinogens.[19] An initial qualitative assessment would be made in accordance with the original categorization system. For each particular substance falling into Category I, an assessment would be made of whether current exposure levels constituted a significant risk. Those chemicals considered to pose such a risk would be regulated down to the lowest feasible level, as before.

Of greater import for the Generic Carcinogen Policy was the presidential inauguration several days later of Ronald Reagan. OSHA's cancer policy was listed by the new Administration's Task Force on Regulatory Relief as one of the first 27 regulations to be reconsidered due to their economic costs. On March 27, 1981, the Generic Carcinogen Policy amendments proposed in January were withdrawn in order to permit OSHA to "address alternatives that had not been fully considered."[20] The policy remained in abeyance from then on. In January of 1982, OSHA officially proposed a partial stay on the publishing of the Candidate and Priority Lists of chemicals[21]; this stay was promulgated a year later.[22]

These actions were largely of symbolic importance, since the agency was not pursuing the hazard identification and regulation provisions of the Generic Carcinogen Policy in any case. In August of 1986, the agency announced its intention to propose rulemaking to revise the standard, but did not follow up with any substantive initiative.[23] In February 1987, the Fifth Circuit Court of Appeals dismissed 16 petitions filed by industry and labor groups concerning various provisions of the Generic Carcinogen Policy on the grounds that nothing had happened in six years and hence that it could be considered dormant.[24] Nevertheless the Generic Carcinogen Policy has remained as OSHA's official policy.[25] The National Institute for Occupational Safety and Health (NIOSH) continues to propose lowest feasible level regulations for substances with laboratory evidence of carcinogenicity, under the authority of the Generic Carcinogen Policy.[26,27] This implies that only a relatively simple administrative decision might be sufficient to revive the policy, a matter of concern for industry groups and the Reagan Administration as the election of 1988 approached. The industry-sponsored American Industrial Health Council and the federal Office of Management and Budget (OMB) both recommended that rulemaking be pursued by OSHA to officially excise the offensive parts of the policy.[28,29] The OMB was particularly harsh on OSHA for allowing the timetable of deregulation to slip, declaring its concern for the Reagan "legacy" and noting that "there is only so much time left." Nevertheless, no action was taken on the policy.

The Generic Carcinogen Policy represents OSHA's most aggressive initiative directed at occupational carcinogens. It embodies all the most conservative, health-protective assumptions about the biological process of carcinogenesis. Of greatest importance, it declares that regulation should proceed for those chemicals for which there exists laboratory but not yet epidemiological evidence of cancer effects. The policy weighs the costs and benefits of regulation in a manner strongly oriented toward regulation. OSHA's definition of "feasible" is a highly stringent one. While not unique in its adoption of this concept of feasibility, the Generic Carcinogen Policy stands out in its effort to expeditiously extend the principle to a sweeping array of chemicals.

Substance-Specific Regulations

OSHA continued to promulgate substance-specific standards in the 1980s, but now with even less success than during the previous decade. The battles of the 1970s had produced three legal and institutional barriers to aggressive regulation of occupational carcinogens which continue to haunt the agency to the present.

- First, the Supreme Court's "significant risk" doctrine underlying the dismissal of the original benzene standard requires the agency to perform a detailed quantitative risk assessment for each candidate carcinogen.
- Second, industry concern over the economic costs of compliance contributed to the development of an increasingly rigorous set of requirements for the agency to estimate the economic impact of each proposed regulation. This requirement for economic studies originated with President Gerald Ford,[30] was extended by

President Jimmy Carter,[31] and culminated with President Reagan's requirement for formal cost-benefit analysis.[32] While OSHA's health standards are officially exempt from these cost-benefit requirements due to a Supreme Court ruling on the cotton dust standard,[33] the agency is required to produce rigorous analyses of the expected cost of compliance with its standards.

- Third, the regulatory strategy of the 1970s raised OSHA to a position of high visibility and vulnerability with respect to the Office of Management and Budget, which has interpreted its role as one of reining in a regulatory process gone amok.[34]

Given these impediments and the general anti-regulatory mood of the Reagan Administration, it is perhaps remarkable that OSHA completed any cancer regulations at all. Its record of achievement during the 1980s was quite modest, consisting of leftover business from the previous decade in the form of the revised benzene[35] and asbestos[36] standards plus the promulgation of two new regulations governing ethylene oxide[37] and formaldehyde.[38]

Two features characterize OSHA's new substance-specific regulations. First, they all cover substances for which there exists epidemiological evidence of carcinogenicity in worker populations. While this might charitably be interpreted as beginning at the top of a priority list similar to those produced by the Generic Carcinogen Policy, it actually constitutes a rejection of the guiding principle of the Generic Carcinogen Policy, namely that regulation should proceed based on animal evidence without waiting for epidemiological studies. The Generic Carcinogen Policy was consciously designed to make the transition from epidemiology to animal laboratory evidence as the basis for OSHA regulations; the similarity between the substance-specific regulations of the 1980s and their counterparts in the 1970s eloquently testifies to the failure of this transition. The substance-specific regulations also symbolize the abandonment of OSHA's efforts to provide leadership for the federal regulatory agencies. This role has now reverted to EPA, which has a carcinogen policy that relies heavily on animal evidence.[39] The second salient feature of the substance-specific regulations is their relative stringency. The permissible exposure limit was reduced by 90 percent in the cases of asbestos and benzene, by 98 percent in the case of ethylene oxide, and by 66 percent in the case of formaldehyde.

The Hazard Communication Standard

The most important standard promulgated by OSHA during the 1980s was the Hazard Communication Standard,[40] which requires labeling of hazardous substances and the development of training programs for workers. While less stringent than the Hazard Identification Standard which was proposed by the Carter Administration[41] and withdrawn in the first months of the Reagan Administration,[42] the Hazard Communication Standard is nevertheless sweeping in scope and potentially significant in impact. Although not focused on carcinogens, the Hazard Communication Standard does impose important new obligations on produces and users of these substances.

The Hazard Communication Standard is officially a "performance standard" which delegates to industry management the right and responsibility to decide which substances are to be included in the labeling and training programs. Nevertheless, the Standard does provide a floor of substances that must be included. This includes chemicals listed as carcinogens by the International Agency for Research on Cancer (IARC) of the World Health Organization[43] plus chemicals included in the National Toxicology Program's (NTP) *Annual Report on Carcinogens*.[44] In addition, chemicals must be designated as potential carcinogens and included in worker training programs if they have produced evidence of carcinogenicity in any well-conducted laboratory studies. According to one estimate, these provisions mandate inclusion of 416 substances based on the IARC and NTP documents plus an additional 2,260 substances with laboratory evidence of carcinogenicity but which are not classified by IARC or NTP.[45] This broad scope of substances immediately evokes the Generic Carcinogen Policy, since it places laboratory data front and center in establishing which substances will be classified as potential occupational carcinogens. Less than 1 percent of these substances have strong epidemiological evidence of carcinogenicity in humans.

While similar to the Generic Carcinogen Policy in terms of scope, the Hazard Communication Standard differs markedly in terms of regulatory requirements. It imposes no permissible exposure limits, much less ones that are the lowest feasible short of ruining the industry. Under the terms of the Standard, chemical manufacturers must prepare summaries of available toxicological and epidemiological information, in the form of a Material Safety Data Sheet (MSDS), and provide these to all purchasers of their products. Manufacturers are also responsible for the labeling of containers of hazardous substances; these labels contain brief warnings and refer to the relevant MSDS. All users of covered substances, not just chemical manufacturers, must develop written hazard communication programs that detail how they will comply with the Standard. These users must also organize worker training sessions which cover the information on the labels and MSDS plus proper handling procedures.

The Hazard Communication Standard provides no direct protections but relies upon worker responses to the new information for its ultimate efficacy. In principle, these responses could occur via turnover[46–48] or collective bargaining.[49–51] While modest in the startup costs it imposes on employers, the Hazard Communication standard has the potential to alter management treatment of suspect carcinogens over the long term. In the short term, however, the Standard's effectiveness will be limited by the poor quality of the MSDS and employer training programs currently in place.[52]

The Air Contaminants Standard

A dramatic departure from substance-specific rulemaking, OSHA's 1989 Air Contaminants Standard[53] imposed permissible exposure limits (PELs) on 164 substances not previously regulated and lowered the PELs for an additional 212 substances that had been regulated under the start-up provisions of the original 1970 Occupational Safety and Health Act. Hailed as the centerpiece of a new approach to standard setting, the Air Contaminants Standard has also been severely critized for the procedure used in selecting chemicals and for its choice of exposure limits.

The Air Contaminants Standard is explicitly based upon the Threshold Limit Values (TLVs) of the American Conference of Governmental Industrial Hygienists (ACGIH), a private non-governmental organization, whereas the 1970 OSH Act designates NIOSH as the primary source for recommended standards. The TLVs and the process by which they are

TABLE 1—Number of Substances Classified as Confirmed or Potential Human Carcinogens by Five Scientific Organizations, and Number of Classified as Carcinogens by OSHA's Four Carcinogen Policies

Total Classified	NIOSH 126	ACGIH 49	NTP 162	EPA 181	IARC 376
Generic Carcinogen Policy	126	49	162	181	376
Substance-Specific Regulations	22	16	19	18	20
Hazard Communication Standard	126	49	162	181	376
Air Contaminants Standard	11	9	6	8	9

TABLE 2—Alternative Risk Management Strategies for Occupational Carcinogens

	Permissible Exposure Limit	Labeling and Worker Training	Exposure and Medical Monitoring
Generic Carcinogen Policy	stringent	yes	yes
Substance-Specific Regulations	stringent	yes	yes
Hazard Communication Standard	none	yes	no
Air Contaminants Standard	weak	no	no

established have come under increased scrutiny, partly as a result of OSHA's reliance upon them. Analysis of the minutes of the TLV committee and other documents has uncovered a major but unacknowledged role played by the producers of the chemicals.[54] An analysis of the epidemiological studies referenced as part of the documentation for the TLVs found that the TLVs were generally established at levels commonly reported as prevalent in industry rather than at levels below which no significant adverse health effects were reported.[55] The TLV-based PELs for 98 substances in the Air Contaminants Standard were considered insufficiently protective by NIOSH.[56] Both industry and labor representatives asserted that the PELs mandated by the Air Contaminants Standard may create some problems of compliance for small firms but none for major producers.[57]

The treatment of occupational carcinogens under the Air Contaminants Standard is inconsistent and incomplete. While 78 of the substances covered by the Standard are considered confirmed or potential human carcinogens by NIOSH, ACGIH, and/or NTP, only 11 are regulated under the standard based on cancer risks. The others are regulated based on noncarcinogenic effects. Excluded altogether from the Air Contaminants Standard are 68 substances considered to be confirmed or potential human carcinogens by NIOSH, ACGIH, and/or NTP.[58]

The Air Contaminants Standard makes no consistent distinction between epidemiological and laboratory evidence in regulating substances as carcinogens. Of the 11 substances regulated as carcinogens, six had epidemiological evidence of cancer effects in workers while five had only laboratory evidence of cancer in animals. Of the 67 substances regulated based on noncarcinogenic effects alone, five were considered confirmed human carcinogens by NIOSH, ACGIH, and/or NTP based on epidemiological evidence. Of the 68 substances considered as confirmed or potential carcinogens by the three scientific bodies but excluded altogether from the standard, 11 had epidemiological evidence of cancer in humans.

The Air Contaminants Standard is also inconsistent in the stringency of the exposure limits placed on particular substances. Of the 11 substances designated as carcinogens, seven have PELs set equal to the TLV, three have PELs set lower than the TLV, and one has a PEL set higher than the TLV. OSHA computed the residual risk of cancer (maximum likelihood estimate) for workers exposed to seven of these 11 substances at the new PELs. These residual cancer risks ranged from 0.3 cases per 1,000 workers to 40 per 1,000, with a median of 3.7 and a mean of 10.1. By way of comparison, the maximum likelihood estimates of the residual risk under the substance-specific standards promulgated during the 1980s ranged from 0.0006 per 1,000 for formaldehyde[59] to 1.7 per 1,000 for ethylene oxide,[60] 6.7 per 1,000 for asbestos,[61] and up to 9.5 per 1,000 for benzene.[62]

Conclusion

OSHA's official cancer policy, the 1980 Generic Carcinogen Policy, is comprehensive in the universe of substances covered and stringent in the exposure limitations proposed, but languishes in ignominious neglect. The substance-specific regulations present a politically risk-averse contrast. Eschewing chemicals lacking epidemiological evidence of cancer in humans, they devote enormous resources to documenting significant risk and economic feasibility and impose permissible exposure limits on a very few substances. The 1983 Hazard Communication Standard rivals the Generic Carcinogen Policy in the scope of substances covered but establishes a quite different and less direct form of incentives for management to reduce workplace exposures. The 1989 Air Contaminants Standard rivals the Generic Carcinogen Policy in imposing permissible exposure limits for hundreds of toxic chemicals, but abandons internal consistency with respect to the stringency of the particular limits imposed and external consistency with the recommendations from scientific bodies.

The inconsistencies between OSHA's four carcinogen policies are highlighted on Tables 1 and 2. Table 1 presents the number of substances commonly found in the US workplace environment that are designated as confirmed or potential human carcinogens by five scientific bodies, plus the number of substances on each of these five lists that is covered by each of OSHA's four carcinogen policies.[53] The breadth of the Generic Carcinogen Policy and the Hazard Communication Standard is illustrated by their coverage of all substances listed by the five scientific bodies. In start contrast, OSHA's substance-specific regulations and the Air Contaminants Standard cover only a very small fraction of the listed carcinogens. Most remarkable, perhaps, is the contrast between cancer designations in the Air Contaminants Standard and the designations by NIOSH, the organization to which OSHA officially responds. NIOSH considers 76 of the substances covered by the Air Contaminants Standard to be potential human carcinogens, but OSHA only set 11 permissible exposure limits based on avoidance of cancer risk. The Standard excludes altogether 26 substances considered occupational carcinogens by NIOSH.

Table 2 describes that risk management strategy embodied in each of OSHA's four carcinogen policies. Two of the policies impose stringent permissible exposure limits, one imposes weak limits, and one imposes none. The encouragement of worker self-help initiatives through container labeling and employee training is comprehensive in three policies but absent from the Air Contaminants Standard. Only the Generic Carcinogen Policy and the substance-specific regulations contain provisions for exposure monitoring and medical surveillance.

Progress in society's efforts to reduce the incidence of work-related cancer will only come as part of a general reform of occupational health policy at OSHA. On the political front, the shortest odds are for continued stalemate and perpetuation of the status quo. Among outside observers of OSHA's policies, however, a consensus may be growing as to a way to save generic rulemaking.[13,64] This would involve a major compromise to give up the illusion of imposing exposure limits at the lowest feasible level, in exchange for the ability to rely upon animal evidence of carcinogenicity and hence accelerate the pace of standard-setting. Briefly, OSHA could focus on a wide range of substances for which the available evidence might not survive a "significant risk" judical review but which could pass a less stringent "material impairment of health" review. For these substances, OSHA could impose moderate "best available technology" standards modeled upon EPA experiences with the Clean Water Act.[65] More stringent (lowest feasible) standards would be reserved for the much smaller class of substances that could withstand a "significant risk" review.

This proposal is certain to elicit criticism both from those dissatisfied with the outcome of technology-based standards for environmental regulation and from those attached to the symbolic value of the "lowest feasible" regulatory approach. Technology-based standards provide no guarantee that the residual risks are acceptably low and may create disincentives for industry to develop new control methods that might then be mandated as the "best available."[66] Nevertheless, the technology-based approach retains its pragmatic appeal and has been incorporated for the control of hazardous air pollutants in the revised Clean Air Act.[67] As for critics insistent on OSHA's lowest feasible level strategy, the fate of OSHA's Generic Carcinogen Policy should serve as a note of caution. The real world of power and politics seems to offer stringent limits and narrow scope, as in the substance-specific standards, or broad scope and weak limits, as in the Air Contaminants Standard. What is needed is a mutually consistent and supportive set of standards that deal with each different aspect of the overall risk management strategy. When combined with the Hazard Communication Standard and the proposed generic exposure monitoring and medical surveillance standards,[68,69] an appropriately revised exposure limit policy could provide the foundations for an effective generic approach to controlling occupational carcinogens. ☐

James C. Robinson, PhD
Dalton G. Paxman, PhD

Acknowledgments

Valuable comments on an earlier draft were provided by William Pease and Robert Spear. Dr. Paxman's work was supported by the Health Effects Component of the University of California Toxic Substances Research and Training Program.

References

1. 45 Federal Register 5002 (1980).
2. 37 Federal Register 11316 (1972).
3. 39 Federal Register 35890 (1974).
4. 39 Federal Register 3756 (1974).
5. 41 Federal Register 46742 (1976).
6. 43 Federal Register 5918 (1978).
7. 43 Federal Register 11514 (1978).
8. 43 Federal Register 19584 (1978).
9. 43 Federal Register 45762 (1978).
10. 7 U.S.C. 135 (1976).
11. 42 U.S.C. 7401 (1976).
12. US Congress, Office of Technology Assessment: Identifying and Regulating Carcinogens. New York: Marcel Dekker Inc, 1989.
13. Mendeloff JM: The Dilemma of Toxic Substances Regulation: How Overregulation Causes Underregulation. Cambridge MA: MIT Press, 1988.
14. McGarity TO: OSHA's Generic Carcinogen Policy: Rule Making under Scientific and Legal Uncertainty. Law Sci Collaboration 1983; 78:55–104.
15. Rushefsky ME: Making Cancer Policy. Albany, NY: State University of New York Press, 1986.
16. Hutt PB, Keller AM: Federal Regulation of Carcinogens in the Workplace: OSHA's Cancer Policy. New York: Harcourt Brace Jovanovich, 1980.
17. Mintz B: OSHA: History, Law, and Policy. Washington DC: Bureau of National Affairs, 1984.
18. *Industrial Union Dep't v. American Petroleum Inst.*, 448 U.S. 607 (1980).
19. 46 Federal Register 4889 (1981).
20. 46 Federal Register 19000 (1981).
21. 47 Federal Register 187 (1982).
22. 48 Federal Register 241 (1983).
23. Occupational Safety and Health Reporter, August 13, 1986, p. 289.
24. Occupational Safety and Health Reporter, February 11, 1987, p. 982.
25. 29 C.F.R. 1900 (1983).
26. Millar JD: Testimony of the National Institute for Occupational Safety and Health on the Occupational Safety and Health Administration's Proposed Rule on Air Contaminants. Presented at OSHA Informal Public Hearing, August 1, 1988. Docket No. H–020.
27. Occupational Safety and Health Reporter: March 28, 1990. p. 1903. (NIOSH recommends exposure to toluene diisocyanate and toluene diamine be reduced to lowest feasible concentration, based on laboratory evidence of carcinogenicity.)
28. Occupational Safety and Health Reporter: December 23, 1987, p. 1207.
29. Occupational Safety and Health Reporter, April 13, 1988, p. 1659.
30. Exec. Order No. 11,821, 3 C.F.R. 926 (1971–75).
31. Exec. Order No. 46,742, 3 C.F.R. 152 (1978).
32. Exec. Order No. 12,291, 3 C.F.R. 127 (1981).
33. *American Textile Manufacturers Institute v. Donovan* 452 U.S. 490 (1981).
34. Olson ED: The Quiet Shift of Power: Office of Management and Budget Supervision of Environmental Protection Agency Rulemaking under Executive Order 12,291. VA J Natural Resources Law 1984; 4:1–80.
35. 52 Federal Register 34460 (1987).
36. 51 Federal Register 22612 (1986).
37. 49 Federal Register 25734 (1984).
38. 52 Federal Register 46168 (1987).
39. 53 Federal Register 32656 (1988).
40. 48 Federal Register 53280 (1983).
41. 46 Federal Register 4412 (1981).
42. 46 Federal Register 12214 (1981).
43. International Agency for Research on Cancer: IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Lyons, France: World Health Organization, Supplement 7, 1987.
44. US Department of Health and Human Services, National Toxicology Program: Fourth Annual Report on Carcinogens. NTP 85–001. 1985.
45. Oleinick A, Fodor WJ, Susselman MM: Risk Management for Hazardous Chemicals: OSHA's Hazard Communication Standard and EPA's Emergency Planning and Community Right-to-Know Regulations. J Legal Med 1988; 9:179–278.
46. Viscusi WK: Job Hazards and Worker Quit Rates: An Analysis of Adaptive Worker Behavior. Int Economic Rev 1979; 20:29–58.
47. Viscusi WK, O'Connor C: Adaptive Responses to Chemical Labeling: Are Workers Bayesian Decision Makers? Am Economic Rev 1984; 74:942–956.
48. Robinson JC: Worker Responses to Work-

place Hazards. J Health Politics, Policy, Law 1987; 12:665–682.
49. Robinson JC: Workplace Hazards and Workers' Desires for Union Representation. J Labor Res 1988; 9:237–249.
50. Robinson JC: Labor Union Involvement in Occupational Safety and Health, 1957–1987. J Health Politics, Policy, Law 1988; 13:453–468.
51. Robinson JC: Worker Responses to Occupational Risk of Cancer. Rev Economics Stat 1990; 77:536–541.
52. Occupational Safety and Health Reporter: November 30, 1988, pp. 1201–1202.
53. 54 Federal Register 2332 (1989).
54. Castleman BI, Ziem GE: Corporate Influence on Threshold Limit Values. *Am J Ind Med* 1988; 13:531–559.
55. Roach SA, Rappaport SM: But They Are Not Thresholds: A Critical Analysis of the Documentation of Threshold Limit Values. Am J Ind Med 1990; 17:727–753.
56. Robinson JC, Paxman DG, Rappaport SM: Implications of OSHA's Reliance upon TLVs in Developing the Air Contaminants Standard. Am J Ind Med 1991; 19:3–13.
57. Occupational Safety and Health Reporter: May 3, 1989. pp. 1965–1967.
58. Paxman DG, Robinson JC: Regulation of Occupational Carcinogens under OSHA's Air Contaminants Standard. *Regulatory Toxicology and Pharmacology*, 1990; 12:296–308.
59. 52 Federal Register 46220 (1987).
60. 53 Federal Register 11416 (1988).
61. 51 Federal Register 22647 (1986).
62. 52 Federal Register 35138 (1987).
63. The figures from the EPA are derived from Methodology for Evaluating Potential Carcinogenicity in Support of Reportable Quantity Adjustments Pursuant to CERCLA Section 102. EPA Pub. No. 600/8–89/53, 1988.
64. Shapiro SA, McGarity TO: Reorienting OSHA: Regulatory Alternatives and Legislative Reform. Yale J Regul 1989; 6:1–63.
65. 33 U.S.C. 307 (1977).
66. McGarity TO: Media Quality, Technology, and Cost-Benefit Balancing Strategies for Health and Environment Regulation. Law Contemporary Problems 1983; 46:159–233.
67. Clean Air Act of 1990, Section 301, Pub. L. 101–549 to be codified as amended at 42 U.S.C. 7412.
68. 53 Federal Register 32591 (1988).
69. 53 Federal Register 32595 (1988).

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This paper was submitted to the journal June 20, 1990, and accepted with revisions January 20, 1991.

Health Care Received by American Children: NCHS Report

The vast majority (83 percent) of American children are covered by health insurance, have a regular source of health care (88 percent), and visited a doctor in the past year for routine health care (64 percent) according to a recent report issued by the National Center for Health Statistics. The report cites new evidence, however, that America's minority and poor children receive less care, including preventive services.

The report, *Health Insurance and Medical Care: Health of Our Nation's Children, United States, 1988*, is derived from data from the 1988 National Health Interview Survey on Child Health, conducted by NCHS. Questions on health insurance and sources of medical care were asked for for all 17,110 children in a nationally representative sample of children 17 years of age and under.

The survey shows that Black newborns and infants were 2 to 3 times more likely to have lacked a routine visit for a check-up or any immunization during the first year of life than were White newborns and infants. Hispanic children were twice as likely to have no health insurance coverage, either public or private, as non-Hispanics.

Most children with a regular source of health care had private care; however, 16 percent of those children (8.7 million) received health care from hospital clinics, emergency rooms, walk-in or emergency centers or other clinics. Clinics were the source of care for 47 percent of Black infants, compared to 16 percent of White infants. For Hispanic infants, almost half received health care in clinics.

The report, *Health Insurance and Medical Care: Health of Our Nation's Children, United States, 1988* is available from the National Center for Health Statistics, Centers for Disease Control, US Public Health Service, 6525 Belcrest Road, Hyattsville, MD 20782; (301) 436-8500.

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American Journal of Public Health

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Postal Information: Second class postage paid at Washington, DC, and additional mailing offices. Postmaster: Send address changes to American Journal of Public Health, 1015 Fifteenth St., NW, Washington, DC 20005. AJPH allotted $40; TNH allotted $6 of APHA membership dues. Information on individual membership appears elsewhere in this Journal, or contact: Director, Membership Services, at APHA headquarters.

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Commentary

How Blind Is Blind Review?

Alfred Yankauer, MD, MPH

ABSTRACT

Background: No representative surveys of scientific opinion about blind review have been published, and there is very little information on the success of the blinding process. The *American Journal of Public Health* has practiced blind review since 1977.

Methods: In 1989 to 1990 312 of its reviewers were asked to identify author and institution in the manuscript they reviewed, to provide clues to such identification, to express their opinion concerning blind review, and to offer reasons for their opinion.

Results: Reviewers claimed to be able to identify author and/or institution in 47% of the 614 chances offered; identification was incorrect 16% of the time, overall identification correct 39% of the time. Self-referencing was the clue to identification in 62%, personal knowledge in 38% of the cases. If only personal knowledge cases are considered, blinding was successful 83% of the time. Blinding was favored by 75% of the reviewers with most asserting it eliminated bias. Reasons given for opposing blind review included the following: blinding not possible, identification will not influence judgment, and its obverse, identification assists judgment.

Conclusions: For the *American Journal of Public Health* blinding is usually, but not always, successful; and the majority of its reviewers favor current policy. Until more definitive data are in, reviewer preference, which differs from journal to journal, seems the most legitimate guide to journal policy on blind review. (*Am J Public Health*. 1991;81:843–845)

> Some judge of authors' names, not words, and then,
> Nor praise nor blame the writings, but the men.—*Alexander Pope*

Introduction

Whether or not to blind reviewers to the identity of the authors whose papers they are asked to review has been hotly debated for at least 20 years. In spite of firmly held opinions pro and con, no representative surveys of scientific opinion have been reported, and there is very little information on the prevalence of blinding as a practice or on the success of the blinding process.

Four surveys of the frequency of blinding have been published,[1–4] but none is based on a random sampling of journals from different scientific disciplines, and their size and response rates often leave something to be desired. Their results suggest that the majority of scientific journals do not practice blind review and that blinding may be more common in the social sciences than in the physical and medical sciences.

Small, narrowly focused studies of reviewer opinion about the desirability of blind review report varying proportions in favor of review: 39% with 11% having no opinion (personal communication, Lew Gidez, FASEB), 41% *(Diabetes Care)*,[5] 49% (37% no opinion, *J Neuropathol-Exp Neurol*),[6] and 89% *(Schol Publ)*.[7]

Personal opinions of the success of blinding not based on actual study also vary: 33%,[8] 50% to 60%,[9] and 90%.[10] The reported success of blinding in *J Neuropathol Exp Neurol* was 66% when 55% of all eligible manuscripts were eliminated because of self-referencing,[6] and 73% *(Arch Intern Med)*[11] when self-referencing was partially eliminated.

Reviewer opinions about signing their reviews will be influenced by journal policy. Some journals have encouraged reviewers to sign their reviews arguing that the review process should be completely open. Journals which encourage the practice report a signing rate of only 43% *(Arch Intern Med)*[11] and 58% *(J Lab Clin Med)*.[12] *Diabetes Care* reported that 80% of its reviewers opposed mandatory signing, but 53% favored optional signing.[5] Two other small reviewer surveys report 77% *(Schol Publ)*[7] and 46%[6] (47% no opinion) favoring anonymity for reviewers.

The editorial board of the *American Journal of Public Health (AJPH)* voted to adopt a policy of blind review in 1976. I was skeptical about its value but agreed to survey reviewer opinion. After the first hundred reviewer returns, it was evident that the vast majority of *AJPH* reviewers favored blind review, and the policy was implemented in 1977. In the 13 following years only two of more than 1000 reviewers have refused to review a manuscript because of the blinding policy.

Contributors to *AJPH* are instructed to submit a second face sheet which includes only the title of the paper. These instructions are usually followed. We remove acknowledgments, but make no further effort to remove identifying page headers (when present contrary to instructions) or to change the text or references. We have been aware that a substantial proportion of our manuscripts are not truly blinded because of text allusions and

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TABLE 1—Proportion of Correct Identifications of Author and Institution According to "Yes" and "Maybe" Responses

	Author		Institution		Total	
Response Type	%	n	%	n	%	n
Yes	96	(67)	89	(87)	92	(154)
Maybe	70	(66)	80	(66)	75	(132)
Total	83	(133)	85	(153)	84	(286)[b]

[a] Difference in proportion: Yes-Maybe total = 17 (95% correct identifications, 25.5, 6.5).
[b] Both chances taken: 112; Author only taken: 24; Institution only taken: 38.

TABLE 2—Proportion of Correct Identifications of Author and Institutions According to Type of Chance Taken

		Correct Identification (%)	
Type of Chance Taken	*N*	Author	Institution
Author only	24	83	—
Institution only	38	—	82
Both author and institution	112	83	87
Total	286[a]	83	85

[a] Both chances taken doubled in total.

self-referencing; we so inform reviewers in a covering letter that accompanies the manuscript.

Because of the current interest in blinding I undertook a more extended study in late 1989 and early 1990. Its purpose was to find out how often blinding was successful, to identify the give-away clues, and to determine the opinions of our reviewers about the policy. Our communications to the reviewer were unchanged except for the addition of a questionnaire.

Methods

A short questionnaire was sent to reviewers with consecutive blinded manuscripts. It asked whether author and institution were identifiable (yes/no/maybe) and if so, to record their names and describe the clues that led to their identification. Identification was considered correct if any one of the several authors or institutions represented were listed in the response. A second set of questions asked whether they favored blind review (yes/no) and their reasons for such an opinion. Reviewers were asked to return the form to the editor with their review. They were requested to complete the form only once although they may have reviewed more than one paper during the period of the study.

One of the arguments against blinding is that the review process should be completely open, i.e., if the identity of authors is disclosed, reviewers, in turn, should be willing to sign their reviews. For this reason, persons who were not in favor of blind review were sent another questionnaire. They were asked whether they would like to receive the full face sheet containing the name and institution of the author on the manuscripts sent to them for review (yes/no/no preference) and, if yes, whether they intended then to sign their reviews (yes/no).

Results

The analyses are based on 305 fully completed and 7 partially completed returns (accounting for slight discrepancies in totals). There were no written refusals although 4 blank questionnaires were returned with a review. There were 78 pairs and three triplets (different reviewers for the same manuscript) among the returns. Thus the data reflect responses of 312 reviewers to 228 manuscripts.

There were responses to 614 potential chances to identify author or institution, 286 (47%) of which were taken. Overall 84% of the responses taken correctly identified author or institution (Table 1). "Maybe" responses were less likely than "yes" responses to be correct, but only 25% of the "maybe" responses were incorrect. Author and institution were identified correctly to the same extent even when responses were disaggregated (Table 2). Thus blinding could be considered successful 53% or 61% of the time, depending on whether successful blinding ignores identification or includes only correct identification.

Among those reviewing the same manuscript there was agreement in 108 (90%) of the 120 chances taken in common; in 11 of the 120 chances the agreement consisted of an incorrect identification by both respondents.

Self-referencing (61.8%) and personal knowledge (38.2%) were the two clues given for identification of author and/or institution. In both cases 16% of the identifications were incorrect. Clues given and incorrect identifications were unrelated to opinions concerning blind review.

Overall, blinding was favored by 75% of the respondents with 8% stating they had no preference. The 17% initially opposed to blind review is reduced to 11%, and the proportion with no preference increased to 14% when the follow-up of those opposed to blind review is considered.

The principal reason given for their opinion by those favoring blind review was the elimination of bias (Table 3). The principal reason given for their opinion by those opposing blind review was failure of the attempt to blind (in 2 out of the 14 chances taken by this group, identification was incorrect). Two other reasons concerned with the effect of knowing author identity on the review itself appear to contradict each other.

Of 30 reviewers initially opposed to blind review who, on follow-up, expressed an interest in receiving the manuscript face sheet, only 8 stated they would sign all their reviews; 19 indicated they would not do so, and 3 would do so only in some cases.

Discussion

Blinding of this group of reviewers was somewhat less successful than has been reported by others,[6,11] perhaps because self-referencing was more common. Moreover, in the type of population studies published by *AJPH,* the text must identify the source of the data. However, self-referencing that yields an unfamiliar name may mean nothing to a reviewer. Many reviewers noted that although self-referencing gave away the name, they were unfamiliar with the authors or their work. If all 82 responses that mentioned only self-reference are eliminated, blinding of author could be considered success-

TABLE 3—Reviewers* Stated Reasons for Opinions About Blind Review

Favor Blind Review	Number
Objectivity: judge content, not author	143
Comfort: less pressure, less hesitation to criticize	14
Helps beginning researcher if name is unknown	12
Total	169
Oppose Blind Review	
Blinding does not work	18
Names do not influence my judgment	15
Knowing author's background and track record contributes to judging paper.	14
Total	47

*Ninety-six respondents (31%) gave no reason for opinion.

ful, whether or not identification was correct, 83% of the time. Many reviewers who favored blind review commented that they could often identify the author.

It is possible that the long-standing blind review policy of the *AJPH* may have influenced the opinion of our reviewers. However, as reported elsewhere,[13] the vast majority of these reviewers also review for many other journals, most of which do not have such a blind review policy.

All reports of the success of manuscript blinding reflect a Hawthorne effect whose impact is unknown: reviewers may seek out author and institutional identity more vigorously than in their usual review because the question is asked. Evidence for this constraint on the interpretation of these data can be found in this study: in 11 of the 120 chances taken in common, 1 reviewer identified author and/or institution correctly, giving as the reason self-reference, whereas the other reviewer of the same manuscript claimed to be unable to make any identification.

Most of those opposed to blind review would not sign their reviews, throwing doubt on the feasibility of a truly "open" review process. As an editor I, too, would be opposed to having the reviewers sign their reviews because their signing would intrude a third person into the decision process. Those who favored blind review were not asked whether they would sign their reviews.

This survey was not designed to assess the effect of blinding on the content of the review or the reviewers' recommendations to the editor. Nevertheless, the findings can be interpreted as evidence both for and against blinding. Against blinding is the fact that in about half the manuscripts some identification was claimed. Thus if identification biases the reviewer, it does so for some authors but not for others, affecting the equity of the review process; an appreciable number of cases may be falsely identified. In favor of blinding is the fact that a small number of reviewers opposed to blind review apparently felt that the track record and background of authors were factors that could influence their judgment about a paper. This may be reasonable for the rare opinion piece, but it implies a possible bias in the case of a research paper that should be judged solely on its merits.

The vast majority of *AJPH* reviewers favoring blind review contrasts with a recent report of a reader survey of the Federation of American Societies for Experimental Biology (FASEB) journal; most of the respondents were also reviewers (Lew Gidez, personal communication). Response rate for this survey was 45%, but there were 871 respondents. Half of the respondents were opposed to blind review compared with 17% or less of the *AJPH* reviewers. One can only speculate about reasons for this difference of opinion; the fact that the *FASEB J* reviewers are closer to the physical sciences and that *AJPH* reviewers are closer to the social sciences may account for some of this difference.

Although blind review may produce a somewhat kinder and more complete review,[11] I doubt that it can remove bias where negative or positive reviewer bias is associated with reviewer knowledge of author identity. I believe one must rely on the reviewers themselves to be frank about their own biases. Many reviewers return papers with a note recusing themselves for a variety of reasons. Moreover, it is the editor, not the reviewer, who makes the decision on a manuscript, and the editor cannot be blinded.

The fact that the majority of *AJPH* reviewers prefer to continue the blind review policy seems sufficient reason to continue it. Until more definitive data are in, reviewer preference, which differs from journal to journal, seems the most legitimate guide to the policy of journals on blind review. □

Acknowledgments

The author thanks the many reviewers who took the time and trouble to respond so fully to the questionnaire they received.

References

1. Crane D. The gatekeepers of science: some factors affecting the selection of articles for scientific journals. Am Sociol 1967; 2:195–201.
2. Coe RK, Weinstock I. Editorial policies of major economic journals. Quart Rev Econ Business 1967; 7:37–43.
3. Cleary JD, Alexander B. Blind review versus non-blind review: survey of selected medical journals. Drug Intell Clin Pharm 1988; 22:601–602.
4. Weller AC. Editorial peer review in U.S. medical journals. JAMA 1990; 263:1344–1347.
5. Service FJ. Manuscript review. Diabetes Care 1983; 6:208–209.
6. Moosy J, Moosy YR. Anonymous authors, anonymous referees: an editorial exploration. J Neuropathol Exp Neurol 1985; 44:225–228.
7. McGiffert M. Is justice blind? An inquiry into peer review. Schol Publ October 1988; 44–48.
8. Armstrong JS. Barriers to scientific communications: the author's formula. In: Harnad S, Peer commentary on peer review. Behav Brain Sciences 1982; 5:197–199.
9. Scarr S. Anosmic peer review: a rose by another name is evidently not a rose. In: Harvard S, Peer commentary on peer review. Behav Brain Sciences 1982; 5:237–238.
10. Morgan PP. Anonymity in medical journals. Can Med Assoc J 1984; 131:1007–1008.
11. McNutt MD, Evans RT, Fletcher RH, Fletcher SW. The effects of blinding on the quality of peer review. JAMA 1990; 263:1371–1376.
12. Knox FG. No unanimity about anonymity. J Lab Clin Med 1961; 97:1–3.
13. Yankauer A. Who are the peer reviewers and how much do they review? JAMA 1990; 263:1338–1340.

Tobacco Smoking as a Risk Factor for Colon Polyps

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ABSTRACT

Background: Data from a cancer screening project among pattern makers were used to evaluate the association between tobacco smoking and prevalence of colon polyps.

Methods: From 1981–1983, 549 White men were examined by flexible sigmoidoscopy and completed self-administered questionnaires including smoking histories.

Results: One or more colon polyps were detected in 76 men. Standardized prevalence rates (SPR) for polyps increased by smoking category (never smoked = 0.094; ex-smokers = 0.118, current smokers = 0.214) and by cigarettes per day, years of smoking, and pack-years among both current and ex-smokers. Both adenomatous and hyperplastic polyps showed an association with smoking while other types of polyps and polyps with unspecified histology did not. The risk associated with smoking was greater for polyps greater than one centimeter in diameter. An interaction with occupational exposures was suggested by a greater increase in the SPR for polyps among current smokers employed as pattern makers for more than 10 years than among current smokers similarly employed for 10 years or less.

Conclusions: Since at least some colon polyps are considered precursor lesions to colon cancer, one of the most common cancers in the United States, this report suggests that the possible link between colon polyps and smoking deserves further evaluation. (*Am J Public Health*. 1991; 81:846–849)

Introduction

Most epidemiologic studies of colon cancer have suggested that there is no strong or moderate elevation in risk of cancer of the colon among smokers.[1,2] Whether there is a slight increase in risk, perhaps limited to a subgroup of susceptible people, is not known, although some recent work suggests this possibility.[3–6] In addition, an association has been noted between smoking and the presence of adenomas and hyperplastic polyps of the colon in men and women.[7,8] Since adenomatous polyps are known to be precursors of colon cancer,[9] the possibility that smoking influences the development of colon polyps deserves further evaluation. The availability of the data from a colon cancer screening project among pattern makers allowed us to evaluate the association between tobacco smoking and prevalence of colon polyps.

Methods

Pattern makers construct prototypes for mass-produced items, such as automobiles and agricultural implements. They can be exposed to wood, metals, fiberglass, plastics, epoxy resins, welding or soldering fumes, cutting oils, solvents, and spray paints. Three partially overlapping epidemiologic studies reported excess colorectal cancer in pattern makers[10–12] and led to cancer screening programs of pattern makers employed by General Motors Corporation and Ford Motor Company[13] and of members of the Pattern Makers League of North America who worked independently or for other corporations.[13] Data from the screening program of the non-General Motors members of the Pattern Makers League employed other than at General Motors Corporation or Ford Motor Company are the subject of this report. The background of this project has been described in more detail elsewhere.[14,15]

From 1981 through 1983, 1,465 White men from 16 factories were examined by flexible sigmoidoscopy and completed a self-administered questionnaire collecting information on sex, age, race, marital status, education, cancer symptoms, respiratory function, medical history, occupational history, and a few dietary constituents including alcohol but not total fat. The participation rate was 48 percent, a low rate due in part to the economic depression that affected the United States automotive industry during the screening program.[12] The unemployment rate among League members rose from 3 percent to 53 percent during the program with many unemployed pattern makers becoming ineligible for the medical insurance coverage that supported the screening.

Men from 11 of the factories (N = 549) also provided smoking histories and form the basis of this report. There is no evidence that the smoking histories, exposures, or polyp rate of the men at the remaining five factories where the workers did not provide smoking histories differed from that at the 11 other factories. The participation rates at the 11 factories where workers provided smoking histo-

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This paper, submitted to the Journal April 6, 1990, was revised and accepted for publication December 20, 1990.

TABLE 1—Characteristics of the Pattern Maker Population

Characteristics	Number	%
Total	549	
Age (years)		
<= 40	191	35
41–55	221	40
56+	137	25
Duration of employment (years)		
<= 10	440	80
11–20	59	11
21+	50	9
Employment Status		
Active	496	90
Inactive	25	5
Retired	15	3
Other	9	2
Unknown	4	1
Diagnosis		
Cancer	3	1
Polyps	76	14
Other gastrointestinal disease	54	10
No gastrointestinal disease	411	75
Unknown	5	1

ries (47 percent) and at the remaining five factories (50 percent) were similar. The age, duration of employment, employment status (e.g. active, inactive, and retired), and screening diagnoses of the study subjects are presented in Table 1.

The screening program identified three men with colon cancer, 76 men with one or more colon polyps, 54 men with other gastrointestinal disease, and 411 men with no evidence of any gastrointestinal disease. Five men had unknown sigmoidoscope results. Thirty-one of the 76 polyp cases had adenomatous polyps, 24 had hyperplastic polyps, and the remainder had other or not otherwise specified polyps.

Cigarettes per day, years of smoking and pack-years were used as indices of exposure to tobacco smoking for each current or ex-smoker. Three men without colon cancer or polyps had unknown smoking histories and were excluded from the analyses. Several subjects had incomplete smoking histories (e.g. ex-smokers with unknown number of cigarettes per day) and were excluded from some analyses when necessary.

We computed standardized prevalence rates (SPRs) for each category of smoking habit, adjusted for age (< = 40 years, 41–55 years, and 56+ years) and alcohol use (daily consumption of beer, wine, or hard liquor: no, yes, unknown), using the age and alcohol distribution of the entire study population participating in the screening as the standard. Smaller categories of age were not necessary for adequate adjustment for age. The measure of association was the risk ratio (RR). Maximum likelihood estimates, adjusted for the age and alcohol distribution of the smokers and the non-smokers among the men from the 11 factories who provided smoking histories, 95 percent confidence intervals (CI), and Chi tests for trend were obtained using EPITOME, personal computer statistical software under development by the Epidemiologic Methods Section, Biometry Branch, of the National Cancer Institute.[16]

Results

The age- and alcohol-adjusted prevalence rate for colon polyps among men who had ever smoked (0.150) was significantly higher than the rate among nonsmokers (0.094) (RR = 1.7, CI = 1.0, 2.9) (Table 2). The prevalence rose by smoking category (ex-smokers = 0.118; current smokers = 0.214; p for trend = 0.008) and by average number of cigarettes per day, years of cigarette smoking, and pack-years among ex-smokers. The increases in cigarettes per day, years, and pack-years of smoking were also significant, but less consistent, among current smokers.

Comparing current smokers to men who never smoked, risks for adenomatous (RR = 2.7, CI = 1.0, 7.1) and hyperplastic polyps (RR = 3.0, CI = 1.0, 8.7) were higher than risk for other or not specified polyps (RR = 1.0, CI = 0.3, 4.1) (Table 3).

The relative risk for smoking also varied by size and number of polyps. Among current smokers, the RR for polyps less than 1.0 centimeter in diameter was 1.5 (CI = 0.7, 3.1) in comparison with a RR of 2.6 (CI = 1.0, 7.0) for larger polyps. Men who never smoked and ex-smokers were at greater risk for only one polyp than for two or more polyps. Among current smokers, the RR was similar for only one (RR = 1.5) and more than one polyp (RR = 1.6).

Among current smokers, risk for polyps was greater in men employed as pattern makers for more than 10 years (RR = 3.0, CI = 1.3, 7.0) than in men employed 10 or fewer years (RR = 1.4, CI = 0.7, 2.9) (Table 4). Greater risks of polyps among the smokers employed more than 10 years were observed at almost all levels of smoking intensity and duration in comparison to the smokers employed 10 or less years. The higher risks in the longer employed workers were not due to a disproportionate number of heavy or long-term smokers in the group in comparison to the smokers employed 10 or less years. There was little difference by duration of employment among men who never smoked or were ex-smokers.

Discussion

The results presented in this report support the previous findings[7,8] of an association between tobacco smoking and risk of colon polyps. Risk increased significantly and consistently with smoking history category, cigarettes per day, years of smoking, pack-years, and diameter of polyps. Smoking increased the risk for hyperplastic and adenomatous polyps, which are considered the primary precursor lesions of colon carcinoma,[1,9,17] more than the risk for other histologic types. The risk of colon polyps among current smokers was greater in men employed as pattern makers more than 10 years than in men employed for shorter periods of time.

Cigarette smoking is known to be strongly associated with coffee consumption[18] and may be associated with other dietary factors, such as fat intake[18] and alcohol consumption,[19–21] which influence the development of colon polyps and cancer. Analyses were adjusted for age and alcohol consumption, but detailed dietary histories were not available in this study to evaluate other possible confounders. Even the adjustment for alcohol, based on only a crude classification, may have been inadequate to completely account for its possible confounding.

The present study was based on prevalent colon polyps detected in a screening program. Because prevalence reflects incidence and duration, it is not clear if the association with smoking is related to duration, rather than incidence, of colon polyps. The increase of risk with the diameter of polyps is consistent with an association with duration; however, differences in growth rates may be responsible. In addition, risk of multiple polyps was not greater than risk of single polyps among smokers.

No information is available on previous colonoscopies among the study subjects; however, the subjects were asked whether a doctor had ever told them they "had a polyp (growth) in the large intestine (colon, rectum, bowel)." The same percentage of smokers (4 percent) and nonsmokers (4 percent) reported a previous diagnosis of polyp. Therefore, there is

TABLE 2—Standardized Prevalence Rates (SPR) and Risk Ratios (RR) for Colon Polyps by Smoking History*

	N	SPR*	RR**	95% CI++
Never smoked	17/185	0.094	1.0	
Ever smoked	59/361	0.150	1.7	1.0, 2.9
Ex-smokers	31/229	0.118	1.4	0.8, 2.5
Cig/day: ≤19	6/74	0.076	0.9	0.4, 2.2
20–39	16/107	0.142	1.5	0.8, 2.9
40+	6/30	0.192	2.5	1.1, 5.8
Test for trend p = 0.0146				
Years of smoking: ≤10	7/92	0.073	1.2	0.5, 2.7
11–25	8/84	0.118	1.1	0.5, 2.4
26+	14/37	0.169	3.0	1.4, 6.4
Test for trend p = 0.0028				
Pack-years: ≤20	9/126	0.069	0.9	0.4, 2.0
21–40	6/42	0.081	2.1	0.9, 4.7
41+	13/43	0.253	3.3	1.6, 6.9
Test for trend p = 0.0013				
Current smokers	28/132	0.214	2.2	1.2, 4.1
Cig/day: ≤19	7/29	0.276	5.5	2.5, 12.1
20–39	12/76	0.146	1.5	0.7, 3.3
40+	7/20	0.330	5.7	2.6, 12.9
Test for trend p = 0.0035				
Years of smoking: ≤10	0/13	0.000	—	
11–25	6/51	0.074	3.3	1.3, 8.3
26+	21/62	0.192	2.8	1.4, 5.5
Test for trend p = 0.0006				
Pack-years: ≤20	3/48	0.069	2.4	0.9, 6.4
21–40	12/41	0.307	4.5	2.2, 9.4
41+	12/41	0.174	2.1	1.0, 4.5
Test for trend p = 0.0014				

*Three subjects with unknown smoking histories were excluded.

+SPR = Standardized prevalence rate, adjusted using the age and alcohol distribution of the entire study population participating in the screening program as the standard population.

**RR = Risk ratio, maximum likelihood estimate, adjusted for the age and alcohol distribution of the smokers and nonsmokers among the men from the 11 factories that provided smoking histories.

++95% confidence interval. Reference category for all comparisons is men who never smoked.

TABLE 3—Rate Ratios (RR) for Colon Polyps by Smoking History and Histologic Type, Diameter, and Number of Polyps

	Never Smoked (185)			Ex-Smoker (229)			Current Smoker (132)		
	N	RR	95% CI*	N	RR	95% CI	N	RR	95% CI
Histology									
Adenomatous	7	1.0		12	1.2	0.5, 2.7	12	2.7	1.0, 7.1
Hyperplastic	5	1.0		7	0.9	0.3, 2.4	12	3.0	1.0, 8.7
Other and NOS	5	1.0		12	1.6	0.6, 4.1	4	1.0	0.3, 4.1
Diameter of Polyp+									
<1 cm	14	1.0		20	1.1	0.5, 2.1	17	1.5	0.7, 3.1
≥1 cm	3	0.7	0.3, 1.9	11	1.0	0.4, 2.4	11	2.6	1.0, 7.0
*Number of Polyps***									
1 only	12	1.0		21	1.3	0.7, 2.6	14	1.5	0.7, 3.3
2 or more	5	0.4	0.1, 1.2	10	0.6	0.2, 1.3	14	1.6	0.7, 3.5

*95% confidence intervals, adjusted for age and alcohol.

+Reference category is the men who never smoked with polyps of less than 1 cm diameter.

**Reference category is the men who never smoked with only one polyp.

no evidence that the nonsmokers had more previous diagnostic tests or polypectomies than the smokers. The lower prevalence of polyps observed in nonsmokers in the current screening program does not appear related to differences by smoking status in participation in previous screening programs.

It is difficult to determine if pattern makers as a group have an increased prevalence of colon polyps in comparison to the general population. There are few published reports on the results of colon screening among asymptomatic subjects using flexible sigmoidoscopy. Most reports are based on subjects presenting with symptoms for a gastroenterology evaluation and, until recently, used the shorter rigid sigmoidoscope. Age-specific prevalence data and information on the size of the polyps detected are necessary for unbiased comparisons since the prevalence of polyps increases dramatically with age and the minimum diameter of reported polyps affects prevalence estimates. An earlier report on this population of pattern makers[14] concluded that the prevalence of polyps was not excessive in comparison with four other asymptomatic populations screened by flexible sigmoidoscopy. Another evaluation of pattern makers,[22] which included some of the same subjects as the Hoar, *et al*, report,[14] concluded there was an excess of polyps in the pattern makers; however, the polyp size may not have been considered adequately.

The observed association between smoking and colon polyps, in particular adenomatous polyps whose progression to cancer has been reported,[9,17,21] suggest that colon cancer may also be affected by use of this product. Colon cancer is not generally considered a tobacco-related malignancy; however, the literature is inconsistent. Most published studies of colon cancer have not provided information on smoking. Slight deficits of colorectal cancer among smokers have been reported in the American Cancer Society survey,[23] the Third National Cancer Survey,[19] and a recent study from Italy.[24] A significant inverse association has been reported among women by Sandler, *et al*,[3] which, they speculate, may be due to an antiestrogenic effect of smoking. On the other hand, Sandler, *et al*,[3] reported an increased rate of colon cancer in male smokers ages 25–44, (nonsmokers and passive smokers: 5.5/100,000 ; ever smokers: 19.0/100,000). Slight excesses of colorectal cancer occurred among male British doctors,[4] US veterans,[5] and, for rectal cancer, female British doctors.[6]

Not all colon polyps result in colon cancer. Although some polyps are considered precancerous lesions, some of them disappeared in a follow-up study.[25] The risk factors for colon cancer and polyps may differ or act with different strength.[26] Smoking may interact with other factors, as suggested in this study by the higher risk of polyps in long-term employees.

TABLE 4—Rate Ratios (RR) for Colon Polyps by Smoking History and Duration of Employment as a Pattern Maker

	Employed ≤10 years			Employed 11+ years		
Smoking History	N	RR	95% CI[†]	N	RR	95% CI
Never smoked	15/144	1.0		2/39	1.1	0.4, 3.0
Ex-smokers	27/183	1.2	0.6, 2.3	4/43	0.9	0.3, 3.0
Current smokers	20/103	1.4	0.7, 2.9	8/26	3.0	1.3, 7.0

[*] Reference category for all comparisons is the men who never smoked and were employed as pattern makers for ≤10 years.
[†] 95% confidence interval, adjusted for age and alcohol.

An explanation for the findings of an association between colon polyps and smoking and the inconsistent relationship between smoking and colon cancer in the published literature is not entirely apparent to us but, may be due to population variations in the distribution of non-, ex-, and current smokers and in the percent of colon cancers with adenomatous polyp precursors. In the current study, there were 185 nonsmokers, 229 ex-smokers, and 132 current smokers whose risk ratios for adenomatous polyps were 1.0, 1.2, and 2.7, respectively. Previous reports in the literature suggest that approximately 30 percent of colon cancers have adenomatous polyp precursors.[27,28] Assuming that this 30 percent has the same smoking distribution and risks as the adenomatous polyp cases and that the remaining 70 percent of colon cancer cases have no risk associated with smoking (risk ratio = 1.0), a weighted overall risk estimate would be 1.15. Slight changes in any of these parameters would result in the inconsistent overall risk estimates for colon cancer and smoking observed in literature, particularly in the presence of residual confounding by dietary and other risk factors. Given the high incidence of colon cancer in the United States, the association deserves further evaluation. ☐

Acknowledgments

The authors acknowledge the cooperation of Charles Romelfanger of the Pattern Makers' League of North America and the efforts of Ki Moon Bang, Vernon McDougall, Knut Ringen, Milagro Rodriguez, and Sandra Tillet of the Workers' Institute of Safety and Health, and Maria Mazorra and Ray Demers of Wayne State University in conducting the screening program of pattern makers which generated the data used in this report. We also thank Patricia Hartge and Robert Hoover of the National Cancer Institute for review of the manuscript.

References

1. Schottenfeld D, Winawer SJ: Large intestine. *In*: Schottenfeld D, Fraumeni JF Jr (eds): Cancer Epidemiology and Prevention. Philadelphia: WB Saunders, 1982; 703–727.
2. US Department of Health, Education and Welfare: Smoking and Health. A report of the Surgeon General. US DHEW Pub. No. 79-50066. Washington, DC: Govt Printing Office, 1979.
3. Sandler RS, Sandler DP, Comstock GW, Helsing KJ, Shore DL: Cigarette smoking and the risk of colorectal cancer in women. JNCI 1988; 80:1329–1333.
4. Doll R, Peto R: Mortality in relation to smoking: Twenty years observation on male British doctors. Br Med J 1976; 2:1525–1536.
5. Kahn HA: The Dorn Study of Smoking and Mortality among US Veterans: Report on eight and one-half years of observation. Natl Cancer Inst Monogr 1966; 19:1–125.
6. Doll R, Gray R, Hafner B, Peto R: Mortality in relation to smoking: 22 years' observations on female British doctors. Br Med J 1980; 280:967–971.
7. Hoff G, Vatn MH, Larsen S: Relationship between tobacco smoking and colorectal polyps. Scand J Gastroenterol 1987; 22:13–16.
8. Kikendall JW, Bowen PE, Burgess MB, Magnetti C, Woodward J, Langenberg P: Cigarettes and alcohol as independent risk factors for colonic adenomas. Gastroenterology 1989; 97:660–664.
9. Buntain WL, Remine WH, Farrow GM: Premalignancy of polyps of the colon. Surg Gynec Obstet 1972; 134:499–508.
10. Robinson C, Waxweiler RJ, McCammon CS: Pattern and model makers' proportionate mortality 1971–78. Am J Ind Med 1980; 1:159–165.
11. Schottenfeld D, Warshauer ME, Zauber AG, *et al*: Study of cancer mortality and incidence in wood shop workers of the General Motors Corporation. New York: Memorial Sloan-Kettering Cancer Center, Department of Epidemiology and Preventive Medicine, 1980.
12. Swanson GM, Belle SH: Cancer morbidity among woodworkers in the US automotive industry. JOM 1982; 24:315–319.
13. Tillett S, Ringen K, Schulte P, McDougall V, Miller K, Samuels S: Interventions in high-risk occupational cohorts: A cross-sectional demonstration project. JOM 1986; 28:719–727.
14. Hoar SK, Bang KM, Tillett S, Rodriguez M, Cantor KP, Blair A: Screening for colorectal cancer and polyps among pattern makers. JOM 1986; 28:704–709.
15. Bang KM, Tillet S, Hoar SK, Blair A, McDougall V: Sensitivity of fecal Hemoccult testing and flexible sigmoidoscopy for colorectal cancer screening. JOM 1986; 28:709–713.
16. Boice JD Jr, Lubin JH, Preston DL: EPITOME: Epidemiologic analysis with a personal computer, programs compiled at the National Cancer Institute Epidemiology and Biostatistics Program, Bethesda, MD, October 1989.
17. Fenoglio CM, Pascal RR: Colorectal adenomas and cancer: Pathologic relationship. Cancer 1982; 50:2601–2608.
18. Schreiber GB, Robins M, Maffeo C, Masters MN, Bond AP, Morganstein D: Confounders contributing to the reported associations of coffee or caffeine with disease. Prev Med 1988; 17:295–309.
19. Williams RR, Horm JW: Association of cancer sites with tobacco and alcohol consumption and socioeconomic status of patients: Interview study from the Third National Cancer Survey. JNCI 1966; 58:525–545.
20. McMichael AJ, Potter JD, Hetzel BS: Time trends in colorectal cancer mortality in relation to food and alcohol consumption: United States, United Kingdom, Australia, and New Zealand. Int J Epidemiol 1979; 8:295–303.
21. Ricker RR, Auerbach O, Garfinkel L, Hammond EC, Frasca JM: Adenomatous lesions of the large bowel: An autopsy survey. Cancer 1979; 43:1847–1857.
22. Demers RY, Demers P, Hoar SK, Deighton K: Prevalence of colorectal polyps among Michigan pattern and model makers. JOM 1985; 27:809–812.
23. Hammond EC: Smoking in relation to death rates of one million men and women. Natl Cancer Inst Monogr 1966; 19:127–204.
24. Ferraroni M, Negri E, La Vecchia C, D'Avanzo B, Franceschi S: Socioeconomic indicators, tobacco and alcohol in the aetiology of digestive tract neoplasms. Int J Epidemiol 1989; 18:556–562.
25. Knoernshild HE: Growth rate and malignant potential of colonic polyps: Early results. Surg Forum 1963; 14:137.
26. Zaridze DG: Environmental epidemiology of bowel cancer. JNCI 1983; 70:389–400.
27. Burt RW: Polyps: An approach to large bowel cancer detection and prevention. Ca Detect Prev 1985; 8:393–398.
28. Clark JC, Collan Y, Eide TJ, Esteve J, Ewen S, Gibbs NM, Jensen OM, Koskela E, MacLennan R, Simpson JG, Stalsberg H, Zaridze DG: Prevalence of polyps in an autopsy series from areas with varying incidence of large bowel cancer. Int J Cancer 1985; 36:179–186.

Passive Smoking During the First Year of Life

ABSTRACT

Background and Purpose: A description of passive smoking during the first year of life might assist planning preventive efforts.

Methods: Changes in the ecology of passive smoking were investigated in a sample of infants in central North Carolina followed from birth to one year of age.

Results: The prevalence of tobacco smoke absorption, indicated by excretion of cotinine, increased from 53 percent to 77 percent (95% CI of difference: 14, 35) during the first year of life. Most infants (92 percent) excreting cotinine at three weeks of age were also excreting it at one year. Moreover, 61 percent of infants not excreting cotinine at age three weeks were excreting it at one year. This increase reflected an increased exposure to household and, particularly, nonhousehold sources of smoke; the proportion of infants exposed to nonhousehold smokers increased from 14 percent to 36 percent.

Conclusions: These findings suggest that prevention of the onset of passive smoking should begin very early. (*Am J Public Health.* 1991; 81:850-853)

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Introduction

Recognition of the adverse health effects of passive smoking in childhood, particularly during the first year of life,[1-3] has led to efforts to reduce exposure to environmental tobacco smoke.[4] To better inform such efforts, we previously reported the frequency of passive smoking and the circumstances in which it occurred in healthy neonates in central North Carolina.[5] The observations from that research were extended by a one year follow-up of a sample of the original study infants. This paper reports the changes in tobacco smoke exposure and absorption during the first year of life, identifies selected correlates of these changes, and considers the implications of findings for prevention of passive smoking during infancy.

Methods

Study Population

The infants were enrolled at birth, from April 1986 to May 1987, in an experimental study of a program to reduce passive smoking and respiratory illness. To be eligible for enrollment, an infant had to have no significant postnatal problems, a birth weight of at least 2,000 gm, and reside in Alamance County or Chatham County in central North Carolina. Infants in the previous neonatal study[5] were recruited from three hospitals where 433 mothers (40 percent of the 1,096 eligible) gave informed consent. Following data collection for those 433 infants, 216 infants began the passive smoking reduction program to which they had been randomly assigned and 217 remained in the control group. Sixty-five infants in the control group were lost to follow-up by one year of age. The remainder of the control group (n = 152) constituted the study sample for the present paper.

The mean number of adults living in study households was 2.3 at both enrollment and at one year. Seventy-five percent of the infants were living in two-parent families as neonates and 70 percent at one year.

Table 1 describes the study sample. There were no significant differences between the 152 infants in the study sample and the remainder of the 1,096 infants who were eligible for enrollment in the gender of the infant, mother's age, mother's race, and smoking by the mother. The study population, however, included significantly more heads of household with more than a high school education (45 percent) than the remainder of the eligible population (34 percent).

Data Collection Procedures

Data collectors visited the homes of enrolled infants when the infants were approximately three weeks of age [mean age (SD): 18 (seven) days, range: nine to 51 days] and before the family's assignment in the intervention trial. The follow-up visit for the data reported in this study was when the infants were approximately one year old [mean age (SD): 12.3 (0.6)

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TABLE 1—Description of Study Sample (N = 152)

Variables	Percent or Mean
Infants	
Female	49%
Male	52%
Mother	
Race	
White	74%
Black	26%
Age	
Mean (SD)	25.6 (5.2) years
Smoking behavior	
Smokers	26%
Mean (SD)[a]	13.7 (8.7) cigarettes per day
Household Heads	
Education	
Less than High School Graduate	18%
High School Graduate only	38%
More than High School Graduate	45%

[a] Smokers only.

months, range: 11.5 to 14.9 months]. Before beginning the interview, the data collector placed a urine collection bag on the infant and explained to the mother that the urine would be analyzed for tobacco smoke products to determine whether the baby had been exposed to smoke. The mother was then asked to describe the smoking habits of all household members and any recent exposure of the infant to tobacco smoke.

Definitions of Variables

An infant was considered to have *contact with smokers* if the infant lived in the same household as a smoker or was in the same place, at the same time, as a nonhousehold smoker, either in the house or elsewhere. A smoker was anyone who smoked at least one cigarette, cigar, or pipeful per day.

Exposure to tobacco smoke was considered to occur when smoke was produced in the infant's presence, that is, in the same room or vehicle as the infant. The amount of exposure was expressed as cigarettes per week smoked in the infant's presence. (See Appendix for method of measurement.)

Smoke absorption was defined as sufficient airway contact with tobacco smoke to result in absorption of nicotine. It was measured by the urinary concentration of cotinine, a major metabolite of nicotine.[2]

Urine cotinine and our questionnaire measure of tobacco smoke exposure are related, but different measures of passive smoking. We have reported analyses of this relationship in infants elsewhere.[5,6] (See Appendix for further discussion of measures of passive smoking.)

Laboratory Procedures

The urine samples were frozen and shipped to the American Health Foundation, in Valhalla, NY. Researchers there analyzed each specimen without knowing the exposure status of the infant.

Cotinine concentration was quantitated by radioimmunoassay with a modification of the method originally described by Langone, *et al.*[7] This protocol uses specific antisera produced by rabbits and has interassay and intraassay variations of 8 percent, with a sensitivity of 1 ng/ml (6 nmol/L). Levels less than 2 ng/ml (12 nmol/L) were reported as below limits of reliable detection.

Creatinine was measured by dry chemistry methods on a Kodak Ektachem 400 analyzer (Eastman Kodak Company, Rochester, NY). The cotinine was divided by the creatinine concentration to adjust the data to reflect the concentration of the urine sample.

TABLE 2—Sources of Exposure to Tobacco Smoke

	Percent of Infants (N = 152)			
Source of Exposure	3 weeks %	1 year %	% Difference (95% CI)	% Change
Any source	39	63	23[a] (14, 32)	+62[a]
Mother	18	29	11 (5, 16)	+61
Nonmaternal household members	24	35	11 (4, 19)	+46
Nonhousehold smokers	14	36	22 (12, 31)	+157
More than one source	15	33	18 (10, 26)	+120

[a] Because of rounding, number does not exactly equal calculations made from table.

Infants of the nine mothers who smoked and breast-fed were eliminated from all analyses that used cotinine as a continuous variable because of the large amount of nicotine and cotinine absorbed through breast-feeding, relative to the amount absorbed through the airways.[8]

Statistical Methods

Distributions of categorical variables were compared by the chi-square statistic or the Fisher's Exact Test. The two-tailed Student t statistic was used to compare means of continuous variables for two groups.

McNemar's test was used to assess changes between three weeks and one year of age in the proportion of infants exposed to tobacco smoke from different sources and the proportion excreting cotinine.

Analyses of cotinine comparing smoke absorption at the two ages involved only the 114 infants who provided urine specimens at both data collection visits.

Results

Exposure to Tobacco Smoke

Between three weeks and one year of age, the proportion of infants reportedly exposed to tobacco smoke from any source increased from 39 percent to 63 percent (Table 2). The proportion of infants exposed to tobacco smoke produced by their mothers, by nonmaternal household members, by nonhousehold smokers, and by more than one of these sources, increased significantly during the first year of life. The greatest increase (157 percent) was in the proportion of infants exposed to smoke produced by nonhousehold smokers. Very similar results were found when the data analyses for Table 2 were repeated for just the 114 infants who provided urine specimens for cotinine analysis.

One infant was in day care at 3 weeks of age. By one year, 90 (58 percent) of the 152 study infants were in day care and 17 (19 percent) of those in day care were exposed there. Only six infants had their only exposure in day care.

The median amount of exposure for the 39 percent of infants who were exposed to any source of smoke at three weeks was 36 cigarettes per week per infant, with a range from two to 250 cigarettes per week. When these exposed three-week-old infants became one-year-olds the median amount of exposure was still 36 and the range was zero to 343 cigarettes per week.

TABLE 3—Distribution of Infants by Smoke Absorption Status at Age One Year Based on Status at Age 3 Weeks (N = 114[a])

	Smoke Absorption Status	
	Age 3 weeks (N)	Age 1 year (N)
Cotinine in urine	+(60)	+(55) 92% − (5) 8%
No cotinine in urine	−(54)	−(21) 39% +(33) 61%

[a] Infants who provided urine samples at both ages.

Among exposed three-week-old infants, the mother contributed, on the average, 34 percent of the cigarettes per week to which an infant was exposed. All household members other than the mother contributed 42 percent and nonhousehold smokers contributed, on the average, 23 percent to the total amount of an infant's exposure. Among exposed one-year-old infants, the mother contributed, on the average, 33 percent; nonmaternal household members, 38 percent; and nonhousehold smokers, 28 percent.

Absorption of Tobacco Smoke

At three weeks of age, 53 percent of the infants had cotinine in their urine. By one year, the proportion excreting cotinine increased to 77 percent (95% CI of difference: 14, 35).

For the infants excreting cotinine at three weeks, the median cotinine concentration was 79 ng/mg of creatinine (51 nmol/mmol) with a range of 9–643 ng/mg (6–413 nmol/mmol). The median among the same infants at one year was 127 ng/mg of creatinine (82 nmol/mmol), range 0–714 ng/mg (0–458 nmol/mmol). Among individual infants, the difference between the urine concentrations at the two ages varied considerably. Although the mean difference was only 11 ng/mg of creatinine (7 nmol/mmol), the 95% CI of the difference was wide: −40 ng/mg (−26 nmol/mmol), 62 ng/mg (40 nmol/mmol).

Table 3 contains the distribution of infants by smoke absorption status at one year, according to smoke absorption status at three weeks. Almost all infants (92 percent) absorbing tobacco smoke at three weeks were also absorbing smoke at one year. In contrast, only 39 percent of infants who were not absorbing smoke at three weeks were still not absorbing smoke at one year. We compared the characteristics of these 21 infants with the 33 infants who also were not absorbing smoke at three weeks, but who became tobacco smoke absorbers by one year. The latter infants were more likely to live in a household with smokers, 30 percent compared with only 5 percent of infants who continued to not absorb smoke at one year. The two groups of infants did not differ by educational level of the head of household, race, or the proportion attending day care at one year.

Discussion

Although passive smoking was already common at three weeks of age, there was a large increase in the prevalence of passive smoking by one year, an increase found in both the proportion of infants exposed to smoke and in the proportion absorbing nicotine. The large number of infants eligible for enrollment who were not in the study sample may limit the generalizability of our results. For example, because the heads of household of infants not included in the study had less education and were therefore more likely to smoke, the extent of passive smoking among study infants may be an underestimate for infants in the entire community.

Our analysis of smoking by household members suggests that the increase in prevalence of passive smoking might be reduced by urging smokers who are already in contact with infants, but not smoking in their presence, to continue to refrain after the newborn period. Considering the large increase during the first year of life in the proportion of infants exposed to smoke produced by nonhousehold smokers, parents should be encouraged to limit contact between their infants and nonhousehold smokers. Mothers may be unaware of all contacts their infants have with smokers, however: 24 percent of the three-week-old and 49 percent of the one-year-old infants in our study were reported to have no contact with smokers but had cotinine in their urine.

Our findings support vigorous early and continued efforts to prevent infant passive smoking. The larger study from which the data reported here are derived is evaluating one such effort.[9] □

Acknowledgments

This research was funded by Grant No. 28895 from the National Heart, Lung, and Blood Institute, National Institutes of Health.

We are grateful to the pediatricians of Alamance County and Chatham County, North Carolina, for supporting our study of their patients. The cooperation of the administrative and nursing staff of Alamance County Hospital, Alamance Memorial Hospital, and North Carolina Memorial Hospital is also greatly appreciated. We thank the staff of the Infant Health Study, whose work made this study possible.

References

1. US Department of Health and Human Services: The Health Consequences of Involuntary Smoking: A report of the Surgeon General. DHHS Pub. No. (CDC) 87-8398. Washington, DC: Govt Printing Office, 1986; 38–49, 58–59, 127–132.
2. National Research Council: Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, DC: National Academy Press, 1986; 137–146, 269–276.
3. Fielding JE, Phenow KJ: Health effects of involuntary smoking. N Engl J Med 1988; 319:1452–1460.
4. Perry CL, Silvis GL: Smoking prevention: Behavioral prescriptions for the pediatrician. Pediatrics 1987; 79:790–799.
5. Greenberg RA, Bauman KE, Glover LH, *et al:* Ecology of passive smoking by young infants. J Pediatr 1989; 114:774–780.
6. Bauman KE, Greenberg RA, Strecher VJ, Haley NJ: A comparison of biochemical and interview measures of the exposure of infants to environmental tobacco smoke. Eval Health Prof 1989; 12:179–191.
7. Langone JJ, Gjika HB, Van Vunakis H: Nicotine and its metabolites: Radioimmunoassays for nicotine and cotinine. Biochemistry 1973; 12:5025–5030.
8. Schwartz-Bickenbach D, Schute-Hobein B, Abt S, Plum C, Nau H: Smoking and passive smoking during pregnancy and early infancy: Effects on birth weight, lactation period, and cotinine concentrations in mother's milk and infant's urine. Toxicol Lett 1987; 35:73–81.
9. Strecher VJ, Bauman KE, Boat B, Fowler MG, Greenberg RA, Stedman H: The development and formative evaluation of a home-based intervention to reduce passive smoking by infants. Health Educ Res 1989; 4:225–232.
10. Coutas DB, Samet JM, McCarthy JF, Spengler JD: A personal monitoring study to assess workplace exposure to environmental tobacco smoke. Am J Public Health 1990; 80:988–990.
11. Benowitz NL, Jacob P: Daily intake of nicotine during cigarette smoking. Clin Pharmacol Ther 1984; 35:499–504.

APPENDIX

Measures of Passive Smoking

Data to measure exposure to tobacco smoke were gathered for both "a typical day last week" and "during the last weekend." The mother identified all the places where the infant spent time, including rooms in the house, at other homes, in cars, and in other places outside the home. For each place, the mother reported all smokers who entered while the infant was there. She also reported whether they smoked, as well as what, and how much they smoked. The amount of exposure was calculated by multiplying the number of cigarettes smoked in the infant's presence during "a typical day" of the previous week by five, and adding the number of cigarettes smoked in the infant's presence during the previous weekend. A cigar or pipeful was considered equivalent to one cigarette. Infants who were cared for more than four hours a week away from their home were defined as in day care. If the mother reported that the day-care workers smoked inside where the children were, then the infants were considered exposed to tobacco smoke. The amount of exposure in day care was not determined.

Both the interview measure of exposure to tobacco smoke and urine cotinine were used as measures of passive smoking because of their different strengths and limitations.[6,10] The interview measure estimates the amount, source, and location of exposure during the previous week, taking into account potentially different smoking habits during the week and the weekend. (We requested a report of exposure during a "typical" weekday because we were interested in a reflection of chronic exposure that was not influenced by unusual experiences during the previous week. However, a possible limitation of using only a "typical" weekday is that exposure in some infants may vary considerably from weekday to weekday.) The questionnaire results are limited by being subject to the memory of the mother and to biased reporting.

The presence of cotinine in urine is an accurate indication that nicotine has been absorbed during the 48 to 72 hours prior to specimen collection, but provides no information about the exposure leading to absorption or about past absorption. The concentration of urine cotinine varies considerably among those exposed to the same amount of nicotine,[11] and the possibility of differences in the same infant at different ages in the pharmacokinetics and metabolism of nicotine remains to be explored. Also, nicotine is only one of thousands of compounds in tobacco smoke and is probably not responsible for the health effects of passive smoking.[1]

Among the study infants who provided urine specimens at three weeks and one year of age, we found the following relationships between exposure to tobacco smoke (cigarettes per week smoked near the infant) and absorption of nicotine (urine cotinine concentration). The Spearman rank correlation at three weeks was 0.50 (95% CI: 0.35, 0.63) and at one year, 0.68 (95% CI: 0.56, 0.77). Among the 60 infants excreting cotinine at three weeks, 60 percent were reportedly exposed during the previous week; whereas among the 54 infants with no cotinine in their urine at three weeks, 20 percent were exposed. Among the 88 infants excreting cotinine at one year of age, 69 percent were exposed during the previous week; whereas among the 26 infants not excreting cotinine at one year, 27 percent were exposed.

Are the Correlates of Cigarette Smoking Initiation Different for Black and White Adolescents?

Sandra W. Headen, PhD, Karl E. Bauman, PhD, Glenn D. Deane, MA, and Gary G. Koch, PhD

ABSTRACT

Background: **Studies of adolescent smoking suggest that the causes of smoking initiation may differ for Blacks and Whites.**

Methods: **Correlates of smoking initiation were examined among 1,277 nonsmokers, ages 12–14, who completed questionnaires in their homes. The analyses examined relationships between smoking initiation and 11 explanatory variables using logistic regression with the combined sample and with Black and White samples.**

Results: **Over two years, 24 percent of Whites and 14 percent of Blacks started to smoke. Whites were more likely to start smoking at age 12 and Blacks at age 14. Having a best friend who smoked increased the odds of initiating smoking over twofold for Whites but had no effect on the odds of smoking for Blacks.**

Conclusions: **Whites initiate smoking earlier than Blacks and are more likely to be influenced by friend behavior. (*Am J Public Health*. 1991; 81:854–858)**

Introduction

Most research on adolescent smoking has involved only White subjects or has not reported findings separately for Blacks and Whites.[1–9] The findings of these studies typically have been applied to both races, but this practice may lead to erroneous conclusions about the behavior of Blacks. The several studies that compared Blacks and Whites found both similar and different correlates of smoking. For example, smoking initiation by both Blacks and Whites has been linked to age, smoking by siblings and friends, perceived availability of cigarettes, intentions to smoke, and ease of refusing cigarettes; risk-taking has been associated with smoking for Blacks but not Whites; parental smoking has been correlated with smoking for Whites but not Blacks.[10–12] Correlates of regular smoking also differed for Black and White samples suggesting that the causes of smoking for these groups differ as well.[13]

This study examines whether the correlates and therefore possible causes of adolescent smoking are the same for Blacks and Whites. The correlates of smoking initiation are first examined for combined samples, testing for the effects of race, then examined separately for Black and White samples.

Methods

Sampling and Survey Procedures

The data are from a field experiment conducted to evaluate the influence of a mass media campaign on the onset of adolescent cigarette smoking. The campaign and its effectiveness are described elsewhere.[14,15] Ten of the 81 Standard Metropolitan Statistical Areas (SMSAs) in the southeastern United States that were similar on social and demographic characteristics and located in nonoverlapping broadcast areas were selected for the experiment. Using cluster sampling procedures, approximately 80 clusters of households were randomly selected in each SMSA to yield an expected average of 224 eligible adolescents per SMSA. Baseline surveys, conducted from April 1 through October 13, 1985, included all eligible adolescents (ages 12–14) within each SMSA (N = 2,534). In the presence of a trained interviewer, adolescents completed self-administered questionnaires in their homes. Follow-up data were collected two years later. For these analyses, data are aggregated for the 10 SMSAs.

Measurement

Cigarette smoking was derived from a 13-category item that ranged from "I have never smoked" to "I smoke two or more packs per day." Adolescents who smoked at least "a few times in the past year" were considered smokers and adolescents who had not smoked in the past year including never smokers were considered nonsmokers. A binary variable rather than the full range was used because the distribution was skewed toward infrequent and light smoking. This defini-

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tion of smoking is consistent with studies that focus on the first or second smoking experience among young adolescents who are experimenting with cigarettes.[4,5] To enhance the validity of self-reports of cigarette smoking, adolescents who completed questionnaires were told that they would be asked to provide aveolar breath and saliva samples to measure actual smoking behavior.[18,19]

Explanatory variables that had been identified as correlates of adolescent smoking in prior research were selected for study.[1,2,4,5,13,18–22] These variables are associated with the social learning and problem behavior theoretical perspectives and are described in the appendix.[7,10,11,21] Age, gender, and parent education were included as control variables. The following transformations were performed:

- Parental education, age, educational intentions, and availability of cigarettes were included as binary variables because the distribution of responses for their original categories were skewed.
- Religiosity, peer versus parental influence, parental permissiveness, and rebelliousness were included as continuous scales and derived by obtaining the means of several items. Closeness to mother and mother's approval of adolescent smoking were included in their original forms.

Analysis

To examine smoking initiation directly, only the 1,277 subjects from the 1985–87 panel who had not smoked within the year preceding 1985 were included in these analyses, eliminating the possible confounding effects of prior smoking behavior on 1987 measures.

Evaluation of correlates of adolescent smoking was primarily based on a logistic regression analysis that included race and 10 other explanatory variables as predictors of smoking initiation. This model was a simplification of another model that had included 15 explanatory variables. Four variables—intentions to smoke, parental permissiveness, mother's approval of adolescent smoking, and availability of cigarettes—were dropped from the earlier model. The −2 Log likelihood statistic showed that the explanatory capacity of the models that included and excluded these variables was similar. The number of subjects who indicated an intention to smoke at baseline was too small for reliable estimation of its effect. The remaining variables were included to examine whether rates of maternal smoking reported by Black adolescents may be associated with social environments characterized by parental leniency, easy access to cigarettes, and tolerance of adolescent smoking. The effects of these variables were expected to be relatively small and essentially no explanatory ability for them was confirmed after adjustment for other variables in the model.

This study reports findings of logistic regression analyses for Black and White samples that resulted from the following procedures. For the combined samples, stepwise procedures were used to identify significant two-way interactions among 11 explanatory variables. Significant interactions with race were of particular interest because of their implication of different relationships for Blacks and Whites. In a subsequent step, three-way interactions, including race and explanatory variables involved in significant two-way interactions, were also examined. Finally, the model that had been obtained on the combined samples was evaluated for the separate samples of Black and White adolescents. The significant interactions between race and the variables, friend smoking and age, in the equation for the combined samples provided the justification for these analyses.

Results

Subject Participation

Of the 2,534 adolescents who were eligible for study, 2,102 participated in 1985 and 1,637 participated in both 1985 and 1987. Among 429 subjects who were eligible but did not participate in 1985, 50.7 percent (n = 218) refused and 49.3 percent (n = 211) had moved from the area, could not be reached after multiple attempts, or were excluded for other reasons. Failure to locate subjects and missing information were the primary reasons for attrition by 1987. Other analyses of these data showed that subjects in the panel and those who failed to complete the follow-up questionnaire did not differ on the following dimensions: age, gender, educational intentions, religiosity, rebelliousness, participation in school activities, and parental closeness.[23] However, subjects lost to follow-up were more likely to be regular smokers at baseline than adolescents who participated at both data collection points (5.6 percent versus 3.1 percent, respectively).[15]

TABLE 1—1987 Smoking Status of 1985 Nonsmokers

Characteristics	Total N	Smoker Percent	Nonsmoker Percent
12 years old			
Female			
Black	63	9.5	90.5
White	130	23.1	76.9
Male			
Black	56	7.1	92.9
White	143	26.6	73.4
Subtotal	392	19.9	80.1
13 years old			
Female			
Black	58	13.8	86.2
White	164	13.4	86.6
Male			
Black	84	11.9	88.1
White	135	24.4	75.6
Subtotal	441	16.6	83.4
14 years old			
Female			
Black	63	20.6	79.4
White	150	26.7	73.3
Male			
Black	67	17.9	82.1
White	142	30.3	69.7
Subtotal	422	25.6	74.4
Total		20.6	79.4
Black	391		
White	864		
Grand Total	1,255		

TABLE 2—Descriptive Statistics for Explanatory Variables (1985)

Variables	Means or Proportions: Black	Means or Proportions: White	Difference (95% CI)
Best friend smokes^{a}	22.90%	26.90%	4.00 (3.94, 4.06)
Mother smokes^{a}	40.70%	27.30%	13.35 (13.32, 13.38)
Religiosity^{b}	2.40	2.30	.10 (.02, .18)
Rebelliousness^{c}	.22	.23	.01 (−.05, .07)
Close to mother^{d}	4.58	4.48	.10 (00, .20)
Peer/parent influence^{e}	3.30	2.60	.70 (.58, .82)
Educational intentions^{f}			
college impossible^{g}	8.20%	2.20%	6.00 (5.96, 6.04)
college possible^{h}	34.80%	24.50%	10.20 (10.10, 10.30)

Categories were:
a) 1 = smoker, 0 = nonsmoker;
b) 3 = religion important, 0 = religion unimportant;
c) 1 = rebellious, 0 = nonrebellious;
d) 5 = extremely close to mother, 1 = not at all close;
e) 4 = parents more important, 0 = peers more important;
f) distributions for reference category (college certain) were 57% and 73.3% for Blacks and Whites respectively;
g) 1 = college impossible, 0 = college certain;
h) 1 = college possible, 0 = college certain.

TABLE 3—Adjusted Odds Ratios (and 95% confidence intervals) for Logistic Regression of Explanatory Variables on Smoking Initiation

Variables	Adjusted Odds Ratios (95% CI): Blacks (n = 401)	Adjusted Odds Ratios (95% CI): Whites (n = 876)
Best friend smokes^{a}	1.05 (.53, 2.10)	2.44 (1.68, 3.53)
Mother smokes^{a}	.01 (.00, .76)	.06 (.01, .59)
Religiosity	.83 (.56, 1.22)	.81 (.65, 1.01)
Rebelliousness	1.34 (.37, 4.85)	2.39 (1.17, 4.85)
Close to mother	.60 (.39, .92)	.76 (.59, .98)
Peer/parent influence	1.04 (.75, 1.45)	.80 (.68, .94)
Educational intentions^{b}		
College impossible	2.10 (.79, 5.58)	1.73 (.60, 5.00)
College possible	1.25 (.63, 2.48)	1.40 (.95, 2.08)
Parent education^{c}		
< High school	.87 (.31, 2.46)	.30 (.12, .75)
College graduate	1.27 (.54, 3.00)	.81 (.55, 1.20)
Age^{d}		
13 years old	1.12 (.40, 3.10)	.51 (.31, .84)
14 years old	2.53 (1.14, 5.64)	.93 (.62, 1.40)
Gender^{e}	1.17 (.58, 2.39)	.59 (.41, .85)
Race^{f}		

High scores mean more of a quality or parents influential. Reference categories were:
a) nonsmoker,
b) college certain,
c) high school graduate,
d) 12 years old,
e) male,
f) white.

Model Chi square = 25.74, df = 16, ($p < .06$), and 100.43, df = 16, ($p < .001$) for Black and White samples respectively.

Racial Differences in Smoking Initiation

Associations among race, gender, and 1987 smoking status of subjects who were nonsmokers in 1985 were examined separately for 12, 13, and 14 year olds. The results, shown in Table 1, indicate that, with the exception of 13 year old females, more White than Black adolescents initiated smoking during this two-year period. Smoking initiation more than doubled with age among Black adolescents, but increased only slightly with age among White adolescents.

Black-White Differences in Explanatory Variables

Results shown in Table 2 reveal some notable differences between Black and White adolescents on the explanatory variables. More Blacks than Whites reported that their mother smokes, but fewer Blacks reported having a best friend who smokes. Also, Black adolescents considered parents more important influences than did White adolescents. The majority of adolescents were confident that college was in their future, although fewer Blacks then Whites thought so. More Blacks than Whites were tentative about college attendance (college possible) or reported that a college education would be impossible for them.

Correlates of Smoking Initiation

The results of logistic regression analyses for Black and White samples reveal the following main effects, shown in Table 3, and several interactions that are discussed later:

- Friend behavior was more strongly related to smoking initiation among White than among Black adolescents; the odds of smoking if a friend smoked were 2.44 times greater among White adolescents but did not increase among Black adolescents.
- The age of smoking initiation also differed for Blacks and Whites; compared with Blacks, Whites were more likely to start smoking at age 12; Blacks were more likely to start smoking at age 14 than at ages 12 or 13.

Significant interactions in these equations revealed several findings. For both Black and White samples the odds of initiating smoking if mother smoked were extremely small among adolescents who reported being distant from mother (.03 and .10 for Blacks and Whites, respectively) and approached unity among adolescents who reported being close to mother (1.02 and .98 for Blacks and Whites, respectively). Another finding was the difference in smoking initiation among White males and females from low versus higher socioeconomic backgrounds. The interaction between gender and parent education in the equation for the White sample indicated that when neither parent graduated high school, males were 3.53 times as likely as females to initiate smoking; when one parent completed high school or college, males were only .58 times as likely to initiate smoking compared with females.

Discussion

The results of this study reveal two important findings regarding racial differences in adolescent smoking initiation. The first is that friend smoking is related to smoking initiation among Whites but not among Blacks. This observation suggests that smoking prevention programs that focus on the attitudes and behaviors of peers may not be effective with Black adolescents.

The second important finding is that Whites initiated smoking earlier than Blacks, an observation that is consistent with results of other studies conducted in the southeastern United States.[10,11,18] This outcome suggests that smoking prevention programs should be implemented in elementary grades to deter smoking among Whites and should continue into middle school to prevent smoking initiation among Blacks.

The results of this study suggest that racial differences in adolescent smoking can be examined more effectively with stratified analyses; aggregating data for Blacks and Whites obscures differences between these groups. For example, equations for both Black and White samples showed that adolescents who felt close to their mothers did not initiate smoking if their mother smoked. Also, the differences in smoking initiation among males and females from low versus higher socioeconomic backgrounds were found for the White sample only.

Our results also show that smoking rates and sample sizes must be sufficiently large to detect meaningful relationships for the Black sample when stratified analysis is used. The small proportion of smokers among Black adolescents in this study (14 percent of Blacks smoked in 1987 compared with 25 percent of Whites) and the smaller size of the Black sample decreased the capacity of logistic regression to explain smoking among Blacks.

In summary, this study found that Black adolescents initiated smoking later than Whites and were not influenced by their friends to start smoking. These findings indicate the need for more research on how differences in correlates of adolescent smoking contribute to differences in smoking among Blacks and Whites. We suggest greater emphasis on identifying and testing models of adolescent smoking that consider distinctive cultural and environmental factors that shape the behavior of Black adolescents. □

Acknowledgments

This research was supported by Grant CA38392 from the National Cancer Institute of the National Institutes of Health. Thanks to Connie Padgett for comments on an earlier draft of this paper.

References

1. Bauman KE, Fisher LA, Bryan ES, Chenoweth RL: Antecedents, subjective expected utility, and behavior: A panel study of adolescent cigarette smoking. Addict Behav 1984;9:121–136.
2. Chassin L, Presson CC, Sherman SJ: Stepping backward in order to step forward: An acquisition-oriented approach to primary prevention. J Consult Clin Psychol 1985;53:612–622.
3. Collins LM, Sussman S, Rauch JM, *et al*: Psychosocial predictors of young adolescent cigarette smoking: A sixteen-month, three-wave longitudinal study. J Appl Soc Psychol 1987;17:554–573.
4. Friedman LS, Lichtenstein E, Biglan A: Smoking onset among teens: An empirical analysis of initial situations. Addict Behav 1985;10:1–13.
5. Hirschman RS, Leventhal H, Glynn K: The development of smoking behavior: Conceptualization and supportive cross-sectional survey data. J Appl Soc Psychol 1984;14:184–206.
6. Johnston LD, O'Malley PM, Bachman JG: Drug use among American high school students, college students, and other young adults: National trends through 1985. Rockville, MD: National Institute on Drug Abuse, 1986.
7. Marcos AC, Bahr SJ, Johnson RE: Test of a bonding/association theory of adolescent drug use. Soc Forces 1986;65:135–161.
8. McAlister AL, Krosnick JA, Milburn MA: Causes of adolescent cigarette smoking: Test of a structural equation model. Soc Psychol Q 1984;47:24–36.
9. Perry CL, Murray DM, Klepp K: Predictors of adolescent smoking and implications for prevention. MMWR (Suppl) 1987;36:41S–45S.
10. Hunter SM, Baugh JG, Webber LS, Sklov MC, Berenson GS: Social learning effects on trial and adoption of cigarette smoking in children: The Bogalusa Heart Study. Prev Med 1982;11:29–42.
11. Hunter SM, Croft JB, Vizelberg IA, Berenson GS: Psychosocial influences on cigarette smoking among youth in a southern community: The Bogalusa Heart Study. MMWR (Suppl) 1987;36:17S–23S.
12. Sussman S, Dent CW, Flay BR, Hansen WB, Johnson CA: Psychosocial predictors of cigarette smoking onset by White, Black, Hispanic, and Asian adolescents in Southern California. MMWR (Suppl) 1987;36:11S–16S.
13. Castro FG, Maddahian E, Newcomb MD, Bentler PM: A multivariate model of the determinants of cigarette smoking among adolescents. J Health Soc Behav 1987;28: 273–289.
14. Bauman KE, Brown JD, Bryan ES, Fisher LA, Padgett CA: Three mass media campaigns to prevent adolescents cigarette smoking. Prev Med 1988;17:510–530.
15. Bauman KE, LaPrelle J, Brown, JD, Koch GG, Padgett CA: The influence of three mass media campaigns on variables related to adolescent cigarette smoking: Results of a field experiment 1990. Am J Public Health 1991;81:597–604.
16. Bauman KE, Dent CW: Influence of an objective measure on self-reports of behavior. J Appl Psychl 1982;67:623–628.
17. Murray DM, O'Connel CM, Schmid LA, Perry CL: The validity of smoking self-reports by adolescents: A reexamination of the bogus pipeline procedure. Addict Behav 1987;12:7–15.
18. Baugh JG, Hunter SM, Webber LS, Berenson GS: Developmental trends of first cigarette smoking experience of children: The Bogalusa Heart Study. Am J Public Health 1982;72:1161–1164.
19. Brunswick AF, Messeri PA: Gender differences in the processes leading to cigarette smoking. J Psychosoc Oncol 1984; 2:49–69.
20. Fishbein M: Social psychological analysis of smoking behavior. *In:* Eisner JR (ed): Social Psychology and Behavioral Medicine. New York: John Wiley, 1982;179–197.
21. Jessor R, Jessor SL: Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth. New York: Academic Press, 1977.
22. Newcomb MD, Maddahian E, Bentler PM: Risk factors for drug use among adolescents: Concurrent and longitudinal analyses. Am J Public Health 1986;76:525–531.
23. Foshee VA: The role of parents in the initiation of adolescent cigarette smoking: An empirical examination of control theory. Dissertation, School of Public Health, University of North Carolina at Chapel Hill, 1990.

APPENDIX

Explanatory Variables

Variable names and categories	Description and original form
Best friend smokes Mother smokes 0 = nonsmoker (category 1) 1 = nonsmoker (categories 2–5)	Cigarettes smoked per day; five-point scale from 1 = none to 5 = more than one pack per day
Intentions to smoke 0 = do not intend to smoke (categories 1–3) 1 = intend to smoke (categories 4, 5)	Probability of smoking in two years; five-point scale from 1 = sure I will not smoke to 5 = sure I will smoke
Availability of cigarettes 0 = hard to get (categories 1, 2) 1 = easy to get (categories 3, 4)	Ease of getting cigarettes; four-point scale from 1 = very hard to 4 = very easy
Mother's approval of adolescent smoking	How mother would feel if adolescent smoked; five-point scale from 1 = strongly disapprove to 5 = strongly approve
Educational intentions 1 0 = college certain (categories 4, 5) 1 = college impossible (category 1)	How likely is college? five-point scale from 1 = attendance, impossible to 5 = attendance certain
Educational intentions 2 0 = college certain (categories 4, 5) 1 = college possible (categories 2, 3)	(same as above)
Religiosity; four-point scale from 0 = religion not important, never go to services to 3 = religion important, services weekly	Derived from mean of two, four-point scales: religion important? 1 = not important to 4 = very important; how often services attended? 1 = never to 4 = once a week or more
Rebelliousness continuous variable; range 0, 1 0 = nonrebellious 1 = rebellious	Derived from mean of four binary items; 1 = yes, 2 = no; try to follow rules? break rules you disagree with? break rules for sake of it? break rule to prove a point?
Close to mother; five-point scale from 1 = not close to 5 = extremely close	Feel close to mother?
Peer/parent influence; five-point scale from 0 = friends more important to 4 = parents more important	Derived from two, five-point scales; who do you listen to when you have problems? who influences your behavior? from 1 = friends more often to 5 = parents more often (3 = friends/parents equally)
Parental permissiveness/independence; continuous scale, range 0, 1	Derived from mean of five binary items; 1 = yes, 2 = no; weekend curfew? choose own friends? choose own clothes? regular bedtime? say parents are strict?
Parent education 1 0 = high school graduate (categories 2, 3, 4) 1 = less than high school (category 1)	Highest grade of either parent; six-point scale from 1 = did not graduate from high school to 6 = more than four years of college
Parent education 2 0 = high school graduate (categories 2, 3, 4) 1 = college graduate or higher (categories 5, 6)	(same as above)

Health Behaviors, Risk Factors, and Health Indicators Associated with Cigarette Use in Mexican Americans: Results from the Hispanic HANES

David J. Lee, PhD, and Kyriakos S. Markides, PhD

ABSTRACT

Background: Cigarette smokers often engage in other, potentially deleterious, health behaviors. Such behaviors have not been well documented in Mexican American smokers.

Methods: Data from the Southwestern sample of the Hispanic Health and Nutrition Examination Survey (HHANES) were employed to investigate differences in health behaviors, risk factors and health indicators between cigarette smokers and nonsmokers among Mexican Americans. Differences between those smoking < 10 and 10 or more cigarettes per day were also examined by age group and gender.

Results: Positive associations between smoking status and heavy coffee and alcohol consumption were found across gender and age groups. Less consistent was the finding that smokers weighed less than nonsmokers. Lower systolic and diastolic blood pressures in middle-aged smokers, and higher levels of depressive symptomatology among smoking women were found. Those smoking 10 or more cigarettes per day were more likely to report heavy coffee consumption, with younger men reporting greater activity limitation due to poor health. Middle-aged men and women in the 10+ category were generally in better health than lighter smokers.

Conclusions: Modest associations between cigarette smoking, health behaviors and risk factors found in other studies were confirmed in this Mexican American population. Few significant associations between smoking and health status were noted. (*Am J Public Health.* 1991;81:859–864)

Introduction

Cigarette smoking is the leading cause of preventable mortality in the United States with approximately 390,000 deaths annually attributed to this health behavior.[1] The prevalence of smoking varies by age, gender, and ethnicity. Males have higher rates of smoking than do women although the gap in rates has narrowed in recent years.[2] Smoking rates climb dramatically in adolescence, peak in middle age, and decline in old age. Overall, smoking rates among Mexican American men are comparable to estimates for Black American males (44 versus 41 percent)[3,4] while rates for Mexican American women are comparable to Caucasian women (25 versus 27 percent).[3,4] However, the prevalence of heavy cigarette consumption is lower among both male and female Mexican Americans than in other ethnic groups.[5]

While much research has focused on basic epidemiologic relationships between smoking and health outcomes, some research has focused on the relation between smoking and other risk factors. The importance of this research is four-fold:

- smoking may interact with other risk factors producing synergistic increases in the risk of negative health outcomes[6];
- smokers attempting to quit often need to reduce exposure to other health behaviors (e.g. alcohol consumption) which prompt smoking behavior;
- smokers who engage in multiple negative health behaviors are less likely to successfully quit smoking[7];
- change in one health behavior may lead to concomitant reductions in other health behaviors.[8,9]

Much of this research has focused on interrelations between alcohol, cigarette, and caffeine use documenting modest associations among these psychoactive substances.[10] Cigarette consumption has also been found to positively correlate with cholesterol levels,[11] but paradoxically is associated with lower blood pressure in adults.[12,13] There is also an inverse relationship between smoking and obesity.[14]

Although cigarette smoking is correlated with a number of deleterious health outcomes,[15] both gender and aging differences can influence these associations.[16] Therefore it is important to examine these differences in different age and gender groups. In addition, because most individuals do not start smoking after the age of 25, associations between smoking status and health may be influenced by two factors: those who quit smoking due to poor health; and selective survival with those most vulnerable to smoking-related mortality dying during middle age.[17] Cross-sectional analyses often reveal that older smokers are in better health than nonsmokers.[18]

The purpose of this study is to examine health behavior, risk factor, and health indicator correlates of smoking in a representative sample of Mexican Americans. Because of important age and gender differences, these associations will be examined separately by gender and age groups.

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Methods

The data employed are from the Hispanic Health and Nutrition Examination Survey (HHANES) conducted during 1982–84 by the National Center for Health Statistics.[19] Our analysis is limited to Mexican American respondents from the southwestern states (Texas, New Mexico, Colorado, Arizona, and California). The HHANES employed a complex multistage sampling design that aimed at obtaining a representative sample of Mexican Americans in the southwest (as well as Cuban Americans in the Miami (Florida) area and Puerto Rican Americans in the New York City area) ages 6 months to 74 years. The sampling design employed assured a 97 percent representation of the Mexican origin population of the southwest as well as 84 percent of all persons of Mexican origin in the United States (for details on sampling procedures see Gonzalez).[20]

Our analysis is limited to persons ages 20 to 74. A total of 3,935 persons in this age group participated in the household interview, with 3,326 participating in the physical examination where data on body weight and height, blood pressure, cholesterol, alcohol consumption, and depression were collected.

Measures

Current smoking status was ascertained in all participants who indicated that they had smoked at least 100 cigarettes in their lifetime. Current smokers were asked how many cigarettes they smoked daily. Responses ranged from less than one to greater than 86 with the actual number recorded between these two extremes. The median number of cigarettes consumed in this sample was used to compare participants whose consumption was "moderate" (<10 cigarettes per day) and "immoderate" (≥10 cigarettes per day).

Measures of health behaviors and risk factors included *Heavy Coffee Consumers* (coded 1 for those consuming 4 or more cups per day, 0 for less than 4, collected during a three-month dietary recall interview); *Current Alcohol Consumer* (coded 1 for current alcohol consumer; 0 for abstainer); *Average Alcohol Consumption* (average number of drinks consumed weekly); *Body Mass Index* [weight (in kilograms) divided by the square of the height of the individual (in meters)[21]]; *Systolic* and *Diastolic Blood Pressure* (based on the average of two blood pressure assessments during the physicians's examination[22]) and serum *Cholesterol* (milligrams per deciliter of blood plasma).

Health indicators included in this analysis were Activity Limitation, Self-Rated Health, Physician's Assessment, and Depression. *Activity Limitation* was based on a series of questions regarding limitation in major activity (e.g. job, housework, etc.) because of an impairment or health problem. In addition to the above, persons 71 to 74 years of age were considered to have activity limitation if they needed the help of others with personal care needs or daily routine activities because of any impairment or health problem, (coded 1 for activity limitation, 0 for other). *Self-Rated Health* was based on a single item asking respondents to rate their health as excellent, very good, good, fair, or poor (coded 1 to 5). The *Physician's Assessment* also graded subjects on the same 1 to 5 scale for an overall assessment of health. *Depression* was measured by the Center for Epidemiologic Studies Depression (CES-D) Scale which has been widely used in community surveys and has been extensively validated,[23,24] including with Hispanic populations.[25,26] Internal consistency reliability for the scale with the current data was in excess of .85. Range of scores on the CES-D is 0–60 with higher scores indicative of greater symptomatology.

Analysis

All analyses were calculated separately for males and females in three age groups: 20–39; 40–59; 60–74 years. Due to small sample sizes examination of the smoking consumption groups (<10; 10+) were not undertaken in the 60–74 age group. Log linear and general linear modeling were used to compare mean and percent differences across smoking categories.[27] When age was significantly different across smoking status or level of consumption within these age and sex categories, age-adjusted means and percents were calculated. For continuously distributed variables this analytic approach paralleled that of multiple classification covariance analysis[28] while for dichotomous dependent variables it parallels binary multiple regression analysis.[29] Both approaches require that the covariant be homogeneous across categories of the independent variable. Significant interactions between the covariate and the factor indicate that age adjustment may produce misleading results. Therefore, interactions between age and smoking status and level of consumption were first run to confirm independence. (See Mendes de Leon and Markides[26] for further details on application of this approach).

Analyses were performed with adjustments for sample weights and design effects. The latter were necessary because the HHANES employed a complex multistage sampling design. Sample weights were normalized by multiplying the number of subjects in each group employed in the analysis with each respondent's weight divided by the sum of the weights for the group. Design effects were estimated using the Taylor Approximation method of the SESUDAAN program and were used to adjust variances in the analyses.[30]

Results

The prevalence of smoking and level of cigarette consumption (<10; 10+) by age group and gender is reported in Table 1. Males were more likely to be smokers ($\chi2 = 89.17$, $p<.001$) and to smoke more cigarettes ($\chi^2 = 7.93$, $p<.01$) than females. Prevalence rates also varied by age group with the highest rates found in the middle aged group for both smoking status ($\chi^2 = 7.11$, $p<.05$) and amount of cigarette consumption ($\chi^2 = 9.11$, $p<.05$). The age group by gender status interactions were not significant indicating that the smokers did not disproportionately fall into specific age group and gender categories.

Table 2 presents frequencies and means for health behaviors, risk factors, and health indicators for young males (ages 20–39). Smokers were more likely to report current alcohol consumption, reported greater consumption of alcohol, were more likely to be heavy coffee consumers, and reported poorer self-rated health. Smokers consuming 10 or more cigarettes per day were more likely to be heavy coffee consumers and reported greater activity limitation than smokers consuming less than 10 cigarettes per day.

Table 3 presents frequencies and means for health behaviors, risk factors, and health indicators for young females. As with young males, female smokers were more likely to report current alcohol consumption and were heavier drinkers. Levels of depressive symptomatology were also significantly higher among smokers than non-smokers.

Females consuming 10 or more cigarettes per day, were significantly older than smokers consuming less than 10 cigarettes per day (29.82 versus 28.22). Therefore, after confirming no age by consumption status interactions, age-adjusted means were calculated. As can be seen in

TABLE 1—Prevalence of Smoking Status and Prevalence of Smokers Consuming 10 or More Cigarettes Per Day by Gender and Age Group

		Percent Smokers			Percent Smoking ≥10 Cigarettes/Day	
	N	Male	Female	N	Male	Female
Age Groups						
20–39	2163	42.5	24.2	704	51.5	46.6
40–60	1313	46.4	27.4	455	60.9	50.3
60–74	457	39.7	19.8	133	74.1	54.0

Note: Main effects for smoking status: Age: $\chi^2 = 7.11$; $p<.05$; Gender: $\chi^2 = 89.17$; $p<.001$; Interaction: $\chi^2 = .11$; p = n.s
Main effects for cigarette consumption: Age: $\chi^2 = 9.11$; $p<.05$; Gender: $\chi^2 = 7.93$, $p<.01$; Interaction: $\chi^2 = 2.02$, p = n.s

TABLE 2—Comparison of Nonsmokers and Smokers and Amount of Cigarette Consumption Across Health Behaviors, Risk Factors, and Health Indicators Among Young Men (ages 20–39)

	Smoking Status		Amount of Smoking	
	Nonsmokers (N = 445–586)	Smokers (N = 319–436)	<10/Day (N = 148–212)	≥10/Day (N = 170–221)
	Mean	Mean	Mean	Mean
Health Behaviors/Risk Factors				
% Heavy Coffee Consumers	3	6*	1	10***
% Current Alcohol Consumers	68	79**	81	78
Average Alcohol Consumption^ (Drinks/Week)	4.4	5.5**	5.4	5.7
Body Mass Index (kg/m²)	26.0	25.3	25.4	25.2
Systolic Blood Pressure (mmHg)	117.6	118.3	118.6	117.8
Diastolic Blood Pressure (mmHg)	76.3	75.8	75.6	76.0
Cholesterol (mg/dl)	194.9	197.2	193.3	200.4
Health Indicators				
% with Activity Limitation	9	9	5	13**
Self-Related Health	2.6	2.9***	3.0	2.8
Physician's Assessment	1.4	1.4	1.4	1.4
CES-D Depression	6.3	7.1	6.8	7.3

Note: *$p<.05$; **$p<.01$; ***$p<.001$
^Based on current consumers only (N = 319 nonsmokers; N = 264 smokers; N = 126 <10 cigarettes/per day; N = 137 10+ cigarettes/per day smokers).

Table 3, there were no significant health behavior, risk factor, or health indicator differences between individuals in the two smoking categories.

Table 4 shows the middle-aged (40–59) male smokers were more likely to report greater levels of alcohol consumption, weighed less, reported poorer self-rated health, and had lower systolic and diastolic blood pressure. Smokers consuming 10 or more cigarettes per day had significantly lower diastolic blood pressure than smokers consuming less than 10 cigarettes per day.

Table 5 presents frequencies and means for health behaviors, risk factors, and health indicators for middle-aged females. Age adjustment of means was necessary for the smoking status analyses after determining that the mean age of the smoking group was significantly higher than the nonsmoking group (49.20 versus 48.00). There were no significant interactions between smoking status and age.

Smokers were more likely to be current alcohol consumers, heavy coffee consumers, weighed less, and had lower diastolic blood pressure. Smokers also reported greater depressive symptomatology than nonsmokers. Smokers consuming 10 or more cigarettes per day weighed less, reported better self-rated health and less activity limitation. Table 6 shows that older (60–74) male smokers were more likely to report current alcohol consumption, were more likely to be heavy coffee consumers, and weighed less. Older female smokers were more likely to report current alcohol consumption and were more likely to be heavy coffee consumers.

Discussion

The most consistent differences between smokers and nonsmokers and between those in the two smoking categories were with alcohol and coffee consumption—findings which are in agreement with studies in other populations.[10] In general, smoking status was negatively associated with BMI, a finding also consistent with previous research.[14] Blood pressure was generally lower in smokers than in nonsmokers, although this association was significant only for middle-aged smoking men and women. More consistent associations between smoking status as well as the presence of a dose-response relationship have been reported in other studies.[12]

No significant differences were found for cholesterol levels across smoking groups. The average reported difference in cholesterol levels in smokers and nonsmokers in other studies is approximately 3 percent.[11] In the present analysis, this difference was exceeded only in comparisons of those consuming 10 or more cigarettes per day with those smoking less than this amount in middle-aged women and young men.

Findings relating to physical and emotional health were equivocal, varying by age group and gender. There was limited support that health was poorer in younger, but not older males. In prospective analyses, Hirdes, *et al.*[18] found that smoking status was associated with future

TABLE 3—Comparison of Nonsmokers and Smokers and Amount of Cigarette Consumption Across Health Behaviors, Risk Factors, and Health Indicators Among Young Women (ages 20–39)

	Smoking Status		Amount of Smoking++	
	Nonsmokers (N = 733–868)	Smokers (N = 229–271)	<10/Day (N = 129–147)	≥10/Day (N = 100–124)
	Mean	Mean	Mean	Mean
Health Behaviors/Risk Factors				
% Heavy Coffee Consumers	2	4	3	6
% Current Alcohol Consumers	28	54***	52	56
Average Alcohol Consumption^ (Drinks/Week)	2.8	3.9***	3.6	4.2
Body Mass Index (kg/m²)	25.9	25.9	26.3	25.4
Systolic Blood Pressure (mmHg)	108.8	109.2	109.1	109.5
Diastolic Blood Pressure (mmHg)	70.6	70.1	70.1	70.3
Cholesterol (mg/dl)	190.9	189.0	188.8	190.0
Health Indicators				
% with Activity Limitation	11	12	15	10
Self-Related Health	2.9	2.9	2.8	3.0
Physician's Assessment	1.4	1.5	1.5	1.5
CES-D Depression	8.8	10.7*	10.8	10.5

Note: *p<.05; ***p<.001
^Based on current consumers only (N = 205 nonsmokers; N = 124 smokers; N = 67 <10 cigarettes/per day; N = 57 10+ cigarettes/per day. ++Age-adjusted means are reported.

TABLE 4—Comparison of Nonsmokers and Smokers and Amount of Cigarette Consumption Across Health Behaviors, Risk Factors, and Health Indicators Among Middle-Aged Men (ages 40–59)

	Smoking Status		Amount of Smoking	
	Nonsmokers (N = 247–304)	Smokers (N = 201–265)	<10/Day (N = 81–103)	≥10/Day (N = 118–159)
	Mean	Mean	Mean	Mean
Health Behaviors/Risk Factors				
% Heavy Coffee Consumers	5	9	5	12
% Current Alcohol Consumers	67	75	80	72
Average Alcohol Consumption^ (Drinks/Week)	3.8	5.1**	5.6	4.7
Body Mass Index (kg/m²)	27.7	26.0***	26.1	26.0
Systolic Blood Pressure (mmHg)	127.1	123.0*	125.7	121.5
Diastolic Blood Pressure (mmHg)	83.3	80.0**	82.6	78.4**
Cholesterol (mg/dl)	221.2	219.1	220.1	218.4
Health Indicators				
% with Activity Limitation	22	22	21	23
Self-Related Health	3.0	3.2*	3.2	3.1
Physician's Assessment	1.8	1.8	1.8	1.8
CES-D Depression	5.0	6.2	7.2	5.5

Note: *p <.05; **p<.01; ***p<.001
^Based on current consumers only (N = 163 nonsmokers; N = 157 smokers; N = 68 <10 cigarettes/per day; N = 89 10+ cigarettes/per day.

risk of disability although this association failed to reach statistical significance. Lack of a strong relationship between morbidity and cigarette consumption in this and other studies may reflect selective survival with the heaviest most vulnerable smokers dropping out of the cohort due to premature mortality.[17]

Our findings have implications for epidemiologic investigations of smoking. Associations between smoking and other health behaviors reinforce the need to control for the effect of these behaviors when examining the impact of smoking on health. Psychological status may also be important to control for in such epidemiologic investigations. Depression, found to be associated with smoking status among women in this study and in previous research,[31,32] is also a risk factor for mortality[33]—particularly among cigarette smokers.[34] Researchers may consider controlling for depression in investigations of smoking and health outcomes as well as exploring possible interactions between smoking and depression.[34,35]

Limitations of this study include potential nonresponse bias and reliance on self-report measures of cigarette consumption. Eighty-seven percent of Mexican Americans approached to participate in the HHANES completed the household interview while 76 percent completed the physician's examination.[36] It should be noted, however, that the weighting procedure incorporated in the present analysis includes adjustment for nonresponse.[36]

Although national estimates of self-reported cigarette consumption are correlated with cigarette sales in the United States (average r = .72),[37] specific analysis of self-report data from the HHANES suggest that underreporting of consumption may have occurred. Based on serum levels of cotinine, a principle metabolite of nicotine, Perez-Stable, *et al*,[38] estimated that 20–25 percent of those reporting 1–9 cigarettes used per day were actually underreporting cigarette consumption, while underreporting estimates for those reporting greater than 10 cigarettes per day ranged from 2–11 percent. These findings suggest that a proportion of subjects in the 1–9 cigarette consumption category in the present analysis may have been misclassified due to underreporting.

In general, this paper confirmed the modest associations found between cigarette smoking and health behaviors and risk factors found in other populations, although these associations did vary by age and gender. Furthermore, few significant associations between smoking and health

TABLE 5—Comparison of Nonsmokers and Smokers and Amount of Cigarette Consumption Across Health Behaviors, Risk Factors, and Health Indicators Among Middle-Aged Women (ages 40–59)

	Smoking Status⁺⁺		Amount of Smoking	
	Nonsmokers (N = 447–546)	Smokers (N = 162–196)	<10/Day (N = 79–96)	≥10/Day (N = 79–97)
	Mean	Mean	Mean	Mean
Health Behaviors/Risk Factors				
% Heavy Coffee Consumers	5	9*	6	14
% Current Alcohol Consumers	21	44*	39	54
Average Alcohol Consumption^ (Drinks/Week)	2.4	3.7	3.0	4.4
Body Mass Index (kg/m^2)	28.7	27.5*	29.0	25.9***
Systolic Blood Pressure (mmHg)	123.0	120.7	119.7	119.7
Diastolic Blood Pressure (mmHg)	77.6	74.8***	75.6	73.9
Cholesterol (mg/dl)	216.4	222.1	215.0	225.8
Health Indicators				
% with Activity Limitation	23	22	26	15*
Self-Related Health	3.3	3.3	3.4	3.0*
Physician's Assessment	1.9	1.8	1.8	1.8
CES-D Depression	8.2	12.8***	12.8	12.4

Note: *p<.05; **p<.01; ***p<.001
^Based on current consumers only (N = 96 nonsmokers; N = 74 smokers; N = 29 <10 cigarettes/per day; N = 44 10+ cigarettes/per day. ⁺⁺Age-adjusted means are reported.

TABLE 6—Comparison of Nonsmokers and Smokers Across Health Behaviors, Risk Factors, and Health Indicators Among Older Men and Women (ages 60–74)⁺⁺

	Smoking Status: Men		Smoking Status: Women	
	Nonsmokers (N = 89–121)	Smokers (N = 62–82)	Nonsmokers (N = 145–202)	Smokers (N = 37–51)
	Mean	Mean	Mean	Mean
Health Behaviors/Risk Factors				
% Heavy Coffee Consumers	0	9***	0	9***
% Current Alcohol Consumers	43	63*	11	30**
Average Alcohol Consumption^ (Drinks/Week)	2.9	4.5	—	—
Body Mass Index (kg/m^2)	26.9	25.5*	28.4	27.3
Systolic Blood Pressure (mmHg)	140.1	135.8	138.5	133.9
Diastolic Blood Pressure (mmHg)	80.2	78.2	77.6	74.6
Cholesterol (mg/dl)	214.2	213.6	234.1	235.2
Health Indicators				
% with Activity Limitation	42	30	37	41
Self-Related Health	3.5	3.4	3.7	3.5
Physician's Assessment	2.1	2.1	2.2	2.1
CES-D Depression	5.6	6.1	8.9	12.0

Note: *p<.05; **p<.01; ***p<.001
^Based on current consumers only (N = 42 nonsmokers; N = 40 smokers).
⁺⁺Examination of differences in amount of cigarette consumption was not undertaken due to small sample size.

status were found. Associations between smoking status and depression were found for women, but not men. These findings suggest that smoking researchers examining Mexican American populations also need to control for relevant risk factors and health behaviors associated with smoking when examining relationships between smoking and health. ☐

Acknowledgments

This work was supported by grant AG06235 from the National Institute on Aging and a grant from the Alcoholic Beverage Medical Research Foundation.

References

1. US Department of Health and Human Services: Reducing the Health Consequences of Smoking—25 years of Progress, Report of the Surgeon General. DHHS Pub. No. (CDC) 89-8411. Washington, DC: Govt Printing Office, 1989.
2. Pierce JP, Fiore MC, Novotny TE, Hatziandreu EJ, Davis RM: Trends in cigarette smoking in the United States: Educational differences are increasing. JAMA 1989; 261:56–60.
3. Escobedo LG, Remington PL: Birth cohort analysis of prevalence of cigarette smoking among Hispanics in the United States. JAMA 1989; 261:66–69.
4. Fiore ML, Novotny TE, Pierce JP, Hatziandreu EJ, Patel KM, Davis RM: Trends in cigarette smoking in the United States: The changing influence of gender and race. JAMA 1989; 261:49–55.
5. Castro FG, Baezconde-Garbanati L, Beltran H: Risk factors for coronary heart disease in Hispanic populations: A review. Hispanic J Behav Sci 1985; 7:153–175.
6. Saracci R: The interactions of tobacco smoking and other agents in cancer etiology. Epidemol Rev 1987; 9:175–193.
7. Coambs RB, Kozlowski LT, Ferrence RG: The future of tobacco use and smoking research. *In:* Ney T, Gale A (eds): Smoking and Human Behavior. Chichester: John Wiley and Sons, 1989; 337–348.
8. Puddey IB, Vandongen R, Beilin LJ, English DR, Ukich AW: The effect of stopping smoking on blood pressure—A controlled trial. J Chronic Dis 1985; 38:483–493.
9. Shephard RJ: Exercise and lifestyle change. Br J Sports Med 1989; 23:11–22.
10. Istvan J, Matarzzo JD: Tobacco, alcohol and caffeine use: A review of their interrelationships. Psychol Bull 1984; 95:301–326.
11. Craig WY, Palomaki GE, Haddow JE: Cigarette smoking and serum lipid and lipoprotein concentrations: An analysis of published data. Br Med J 1989; 298:784–788.
12. Green MS, Jucha E, Luz Y: Blood pressure in smokers and nonsmokers: Epidemiologic findings. Am Heart J 1986; 111:932–940.
13. Benowitz NL, Sharp DS: Inverse relation between serum cotinine concentration and blood pressure in cigarette smokers. Circulation 1989; 80:1309–1312.
14. Klesges RC, Meyers AW, Klesges LM, LaVasque ME: Smoking, body weight,

and their effects on smoking behavior: A comprehensive review of the literature. Psychol Bull 1989; 106:204–230.

15. McGinnis JM, Shopland D, Brown C: Tobacco and health: Trends in smoking and smokeless tobacco consumption in the United States. Annu Rev Public Health 1987; 8:441–467.
16. Waldron I: The contribution of smoking to sex differences in mortality. Public Health Rep 1986; 101:163–173.
17. Manton KG, Poss SS, Wing S: The black/white mortality crossover: Investigation from the perspective of the components of aging. Gerontologist 1979; 19:291–300.
18. Hirdes JP, Brown KS, Vigoda DS, Forbes WF, Crawford L: Health effects of cigarette smoking: Data from the Ontario Longitudinal Study on Aging. Can J Public Health 1987; 78:13–17.
19. National Center for Health Statistics: Plan and Operation of the Hispanic Health and Nutrition Examination Survey, 1982–84. Vital and Health Statistics, Series 1, No. 19. DHHS pub. no. (PHS) 85–1321. Public Health Service. Washington, DC: Govt Printing Office, 1985.
20. Gonzalez JF, Ezzati T, White AA, *et al:* Sample design and estimation procedures. *In:* Plan and Operation of the Hispanic Health and Nutrition Examination Survey, 1982–84, National Center for Health Statistics. Vital and Health Statistics, Series 1, No. 19, DHHS Pub. No. (PHS) 85–1321. Washington, DC: Govt Printing Office, 1985.
21. Khosla T, Lowe CR: Indices of obesity derived from body weight and height. Br J Prev Soc Med 1967; 21:122–128.
22. Pappas G, Gergen PJ, Carroll M: Hypertension prevalence and the status of awareness, treatment and control in the Hispanic Health and Nutrition Examination Survey (HHANES), 1982–84. Am J Public Health 1990; 80:1431–1436.
23. Radloff L: The CES-D scale: A self-report scale for research in the general population. Appl Psychol Meas 1977; 8:385–401.
24. Weissman M, Sholomskas D, Pottenger M, *et al:* Assessing depressive symptoms in five psychiatric populations: A validation study. Am J Epidemiol 1977 106:203–214.
25. Roberts RE: Reliability of the CES-D scale in different ethnic contexts. Psychiatry Res 1980; 2:125–34.
26. Mendes de Leon CF, Markides KS: Depressive symptoms among Mexican Americans: A three-generation study. Am J Epidemiol 1988; 127:150–160.
27. SAS Institute Inc: SAS/STAT Guide for Personal Computers, Version 6 Edition. Cary, NC: SAS Institute, 1987.
28. Nie NM, Hull CM, Jenkins JG, *et al:* Statistical Package for the Social Sciences. New York: McGraw-Hill, 1975.
29. Feldstein MS: A binary variable multiple regression method of analyzing factors affecting perinatal mortality and other outcomes of pregnancy. J Royal Stat Soc [A] 1966; 129:61–73.
30. Shaw BV: SESUDAAN: Standard Errors Program for Computing Standardized Rates from Sample Survey Data. RTI/5250/00-01S. Research Triangle Park, NC: Research Triangle Institute, 1981.
31. Chetwynd J: Some characteristics of women smokers. NZ Med J 1986; 99:14–17.
32. Fredrick T, Frerichs RR, Clark VA: Personal health habits and symptoms of depression at the community level. Prev Med 1988; 17:173–182.
33. Friedman HS, Booth-Kewley S: The disease prone personality. A meta-analytic view of the construct. Am Psychol 1987; 42:539–555.
34. Linkins RW, Comstock GW: Depressed mood and the development of cancer. Am J Epidemiol 1990; 132:962–972.
35. Eysenck HJ: The respective importance of personality, cigarette smoking and interaction effects for the genesis of cancer and coronary heart disease. Person Individ Diff 1988; 9:453–464.
36. Delgado JL, Johnson CL, Roy I, Trevino FM: Hispanic Health and Nutrition Examination Survey: Methodological considerations. Am J Public Health 1990; 80(Suppl):6–10.
37. Hatziandreu EJ, Pierce JP, Fiore MC, Grise V, Novotny TE, Davis RM: The reliability of self-reported cigarette consumption in the United States. Am J Public Health 1990; 80:1057–1061.
38. Perez-Stable EJ, Marin BV, Marin G, Brody DJ, Benowitz NL: Apparent underreporting of cigarette consumption among Mexican American smokers. Am J Public Health 1990; 80:1057–1061.

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Standardized Comparisons of the Use of Alcohol, Drugs, and Cigarettes Among Military Personnel and Civilians

Robert M. Bray, PhD, Mary Ellen Marsden, PhD, and Michael R. Peterson, DrPH

ABSTRACT

Background: Understanding the effectiveness of policies and programs aimed at combating substance abuse in the military requires comparison with the civilian population from which military personnel are drawn.

Methods: Standardized comparisons of the use of alcohol, drugs, and cigarettes among military personnel and civilians were conducted with data from the 1985 Worldwide Survey of Alcohol and Nonmedical Drug Use among Military Personnel and the 1985 National Household Survey on Drug Abuse. The two data sets were equated for age and geographic location of respondents, and civilian substance use rates were standardized to reflect the sociodemographic distribution of the military.

Results: Military personnel were significantly less likely than civilians to use drugs, but were significantly more likely to use alcohol and cigarettes and to engage in heavy use of alcohol and cigarettes. Heavy drinking was especially likely among young military men. Military women were similar to military men in their smoking and drug use patterns.

Conclusions: Findings suggest that military policies and programs have been notably effective in reducing drug use, but that efforts to limit alcohol and cigarette use should be intensified. Military efforts directed against alcohol abuse should be targeted toward younger men, while smoking and drug prevention programs should be directed toward both men and women. (*Am J Public Health.* 1991;81:865–869)

Introduction

Alcohol misuse, nonmedical drug use, and cigarette use can have negative effects on the health, social life, family relationships, and work performance of users. Although these negative effects can occur within all segments of society, they may be especially serious for United States military personnel who have the unique mission to protect and defend the nation. Because military personnel are drawn from the civilian population, other things being equal, the prevalence and level of use of alcohol, drugs, and cigarettes of the two populations are expected to be similar. Certain differences between military and civilian life, however, may create substance use rates that differ. It has been argued that distinctive military conditions such as relocation overseas, separation from family, or a greater perceived acceptance of use may foster higher rates of use among military personnel than civilians.[1,2] Alternatively, military policies or programs directed toward the elimination of abuse, such as the adoption of a policy of zero tolerance toward drug use and the implementation of an extensive urinalysis screening program, may foster rates of use among military personnel that are substantially lower than among civilians.[3]

Direct comparisons of differences in rates of use among military personnel and civilians may not be meaningful because the sociodemographic compositions of the two populations differ. Military personnel are disproportionately young and male, characteristics generally associated with higher levels of use.[4] Unstandardized rates may not therefore provide adequate comparisons of the extent of differences in substance use between military personnel and civilians.

Several standardized comparisons of alcohol and drug use among military personnel and civilians have been conducted. Observed differences in rates of problem drinking between military personnel and civilians ages 21 to 59 in the late 1970s, for example, were found to be largely but not entirely accounted for by differences in the demographic compositions of military and civilian populations.[5,6] The prevalence of use of alcohol and drugs among military personnel and for civilians ages 18 to 25 was quite similar in 1980,[7] but in 1982 the prevalence of alcohol use was higher and the prevalence of marijuana and cocaine use was significantly lower for military than civilian men ages 18 to 25.[8]

These comparisons are somewhat limited in that they rest largely on measures of the prevalence of alcohol use that are inadequate to describe the quantity and frequency of use, and comparisons of drug use that are based only on the prevalence of use of specific drugs. Further, some comparisons have been based on civilian data that were collected at a different time than the military data,[7] and other studies compared populations that differed in important ways. Some studies, for example, considered both males and females while others considered only males; some considered only ages 18 to 25 while others con-

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Editor's Note: See also related editorial p 837 this issue.

TABLE 1—Sociodemographic Characteristics of Military Personnel and Civilians, Aged 18–55

Characteristics	Comparison Population	
	Civilian (N = 4,894)	Military (N = 8,084)
Ages (years)		
18–25	25.9	49.3
26–34	28.7	32.3
35–55	45.4	18.4
Sex		
Male	49.8	90.6
Female	51.0	9.4
Race/Ethnicity		
White	79.8	72.6
Black	10.9	16.8
Hispanic	6.9	6.3
Other	2.5	4.3
Education		
Less than high school	17.8	1.3
High school grad/GED	38.6	44.6
Some college	21.6	36.0
College degree or beyond	22.0	18.1

Note: Estimates are unstandardized percentages. Ns (in parentheses) show the number of cases on which weighted data are based.
Civilian data source: 1985 National Household Survey on Drug Abuse.
Military data source: 1985 Worldwide Survey.

sidered a broader age range. No recent military/civilian comparisons of alcohol and drug use are available, and no standardized comparisons of cigarette use have been conducted. For these reasons, general conclusions about military/civilian differences cannot be drawn.

This paper reports results of standardized comparisons of alcohol, drug, and cigarette use among military personnel and civilians in 1985, the most recent date for which comparable data were available. Unlike earlier studies, findings are reported for a broad age range as well as for males and females.

Methods

Data Sources and Analysis Files

Data for military personnel were drawn from the 1985 Worldwide Survey of Alcohol and Nonmedical Drug Use among Military Personnel, sponsored by the US Department of Defense (DoD) and conducted by Research Triangle Institute.[*] Data on civilians were drawn from the 1985 National Household Survey on Drug Abuse (NHSDA), sponsored by the National Institute on Drug Abuse and conducted by the Institute for Survey Research at Temple University and the Department of Sociology at the University of Kentucky.[9] The two surveys differ in overall questionnaire content, order of item presentation, and field procedures. The 1985 Worldwide Survey, for example, was based on self-report questionnaires administered in group settings, while the 1985 NHSDA data were collected in face-to-face interviews. These differences suggest some caution in making comparisons between the two surveys, but both surveys were carefully conducted and are based on large probability samples.

The eligible population for the 1985 Worldwide Survey consisted of all US active-duty military personnel stationed across the world except recruits, service academy students, persons absent without leave, and persons who had a permanent change of station at the time of data collection. A probability sample was selected for the survey using a deeply stratified, two-phase design. Completed questionnaires were obtained from 17,328 active-duty military personnel for a response rate of 80 percent. Of the responses, 85 percent were obtained in group administrations of the survey at military installations; the remainder were obtained by mailing questionnaires to a subsample of nonrespondents from the group administrations. Estimates of the sociodemographic characteristics of the respondent population showed a close correspondence to sociodemographic characteristics of the actual DoD population values.

The target population for the 1985 NHSDA civilian survey consisted of the household population of the continental United States, ages 12 and older. The household population was sampled using a multistage area probability design with 100 primary sampling units. Individuals in group quarters (military installations, correctional institutions, college dormitories, and hospitals) and those who had no permanent residence (homeless people and residents of single rooms in hotels) were not included in the study. The survey was administered face-to-face, and 8,038 completed interviews were obtained for a response rate of 83 percent.

Before analyzing the data, adjustments to the two data sets were made for differences in residential location and age to ensure greater comparability. To equate for location, military data were limited to respondents residing in the continental United States. Active-duty military personnel living off base in civilian housing were excluded from the NHSDA. To equate for age, both data sets were limited to respondents who were ages 18 to 55 (99.8 percent of the military respondents were in this age range). The adjusted data file for military personnel consisted of 8,084 cases, and the adjusted data file for civilians was 4,894 cases.

Standardization Procedures

As shown in Table 1, the military population is younger, somewhat better educated, and more likely to be male or Black than the civilian population.

Direct standardization[10] was used for comparisons of military personnel and civilians. Civilian data were standardized to the joint distribution in the military of age (18 to 21, 22 to 25, 26 to 34, 35 to 55), race/ethnicity (White, Black, other), education (high school diplomas or less, more than high school), and sex. The standardized estimate for the civilian population is interpreted as the mean that would have been obtained if the civilian population had the same sociodemographic distribution as the military.

Both the Worldwide Survey and the NHSDA used complex, nested sampling designs. The estimates and the standard errors of the standardized civilian data and the military data were computed using

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[*] Survey methodology and survey findings for the 1985 Worldwide Survey are described in detail in Bray RM, Marsden ME, Guess LL, Wheeless SC, Pate DK, Dunteman GH, Iannacchione VG: 1985 worldwide survey of alcohol and nonmedical drug use among military personnel. Report prepared for the Assistant Secretary of Defense (Health Affairs), by Research Triangle Institute, Research Triangle Park, NC, 1986.

TABLE 2—Standardized Comparisons of Alcohol Use in Past 30 Days Among Military Personnel and Civilians, by Age Group

	Age Group/Population					
	18–25 years		26–55 years		Total	
Group/Measure	Civilian	Military	Civilian	Military	Civilian	Military
Males	(N = 776)	(N = 1,645)	(N = 1,250)	(N = 5,885)	(N = 2,026)	(N = 7,530)
Any drinking	76.9	87.8***	78.7	81.6	77.8	84.6***
Heavy drinking	14.1	32.1***	9.6	12.9**	11.8	22.3***
Females	(N = 994)	(N = 210)	(N = 1,773)	(N = 334)	(N = 2,767)	(N = 554)
Any drinking	63.2	83.6***	63.9	73.7	63.5	79.0***
Heavy drinking	4.7	7.6	1.7	4.9	3.3	6.3
Total	(N = 1,770)	(N = 1,855)	(N = 3,023)	(N = 6,219)	(N = 4,793)	(N = 8,084)
Any drinking	75.5	87.3***	77.5	80.9*	76.5	84.1***
Heavy drinking	13.1	29.6***	8.9	12.2**	11.0	20.8***

Note: Estimates are percentages. Ns (in parentheses) show the number of cases on which weighted data are based. Civilian data have been standardized to the military data by age, education, race/ethnicity, and sex.

*Different from civilian at the .05 level.

**Different from civilian at the .01 level.

***Different from civilian at the .001 level.

Civilian data source: 1985 National Household Survey on Drug Abuse.

Military data source: 1985 Worldwide Survey.

RATIO2, the software package developed by Research Triangle Institute to analyze surveys with complex sampling designs.** T-tests were used to assess the significance of the differences between the standardized rate for the civilian population and the unstandardized rate for the military. Some of the substantial differences between military personnel and civilians have p values greater than 0.05 because of the relatively large standard errors.

Comparisons are reported for males, females, and both sexes combined and for those ages 18 to 25, 26 to 55, and the total group ages 18 to 55. Comparisons for the younger age group provide information about substance use among those at highest risk for use. For the military, this younger group consists largely of enlisted military personnel. The older age group is generally at lower risk, and in the military this group includes a higher proportion of officers.

Measures of Substance Use

The measures of alcohol use are the prevalence of any drinking and heavy drinking during the past 30 days. Any drinking refers to having had one or more drinks of beer, wine, or liquor during the past 30 days. Heavy drinking refers to consuming five or more drinks per typical drinking occasion at least once a week and is based on a drinking-level classification scheme adapted from Mulford and Miller.[11] The measures of drug use are the prevalence of use in the past 30 days of marijuana/hashish, cocaine, psychotherapeutic drugs (stimulants, sedatives, tranquilizers, analgesics), any drug, and any drug except marijuana. "Any drug" includes marijuana or hashish, inhalants, hallucinogens, cocaine, heroin, and psychotherapeutic drugs. The measures of cigarette smoking are the prevalence of any smoking (one or more cigarettes) and heavy smoking (smoking one or more packs of cigarettes per day) during the past 30 days.

Results

Alcohol Use

Table 2 presents standardized comparisons of the prevalence of any drinking and heavy drinking among military personnel and civilians by age group. Overall, military personnel are substantially more likely than civilians to drink and to drink heavily. This pattern of differences is consistent for males and females and for younger and older age groups, although in some cases differences have p values greater than 0.05. The prevalence of any drinking among all military personnel is about 8 percentage points higher than among all civilians, and the prevalence of heavy drinking among all military personnel is about 10 percentage points higher than that among all civilians.

Military/civilian differences in *any drinking* are more pronounced among women than among men. The rate for any drinking for women in the military is about 15 percentage points higher than the rate for civilian women; for men, the difference in military and civilian rates is about 7 percentage points. Younger military women are almost as likely as military men to drink and more likely than civilian men to do so.

Military men and women are about twice as likely as civilian men and women to be *heavy drinkers*. About one-third of younger military men drink heavily, a rate that is substantially higher than for any other group.

Drug Use

The prevalence of any drug use is substantially lower among military personnel than among civilians, as shown in Table 3. The rate of any drug use among all military personnel is about one-third that among all civilians, 8.4 percent versus 24.1 percent. Similarly, the rate of any drug use among military men is about one-third that among civilian men, whereas the rate of use among military women is about half that among civilian women. These tendencies for any drug use generally hold for specific drugs. Drug use among military women is about the same as among military men, whereas use among civilian women is substantially lower than among civilian men.

For most measures of drug use, the prevalence of any drug use is highest among civilian men, followed by civilian women, military men, and military women. The data in Table 3 also show

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**Wheeless SC, Shah BV: RATIO2 and RATIO3. Research Triangle Park, NC: Research Triangle Institute, 1982, unpublished paper.

TABLE 3—Standardized Comparisons of Drug Use in Past 30 Days Among Military Personnel and Civilians, by Age Group

	Age Group/Population					
	18–25 years		26–55 years		Total	
Group/Measure	Civilian	Military	Civilian	Military	Civilian	Military
Males	(N = 794)	(N = 1,641)	(N = 1,279)	(N = 5,877)	(N = 2,073)	(N = 7,518)
Marijuana	27.3	11.4***	16.9	1.8***	22.0	6.5***
Cocaine	9.7	4.6**	6.0	0.6***	7.8	2.6***
Psychotherapeutics[a]	8.9	4.8*	3.8	1.7*	6.3	3.2**
Any drug[b]	30.0	14.1***	20.2	3.3***	25.0	8.6***
Any drug except marijuana[c]	15.3	8.1**	10.1	2.2***	12.7	5.1***
Females	(N = 1,008)	(N = 210)	(N = 1,803)	(N = 343)	(N = 2,811)	(N = 553)
Marijuana	16.5	3.8***	6.3	1.3***	11.8	2.6***
Cocaine	6.1	3.3	1.8	0.3*	4.1	1.9
Psychotherapeutics[a]	3.6	6.1	3.3	2.7	3.5	4.5
Any drug[b]	19.9	10.3***	9.4	3.7**	15.0	7.2***
Any drug except marijuana[c]	9.2	8.3	5.1	2.7	7.3	5.7
Total	(N = 1,802)	(N = 1,851)	(N = 3,082)	(N = 6,220)	(N = 4,884)	(N = 8,071)
Marijuana	26.2	10.6***	16.0	1.8***	21.0	6.1***
Cocaine	9.3	4.5**	5.6	0.6***	7.5	2.5***
Psychotherapeutics[a]	8.3	4.9*	3.8	1.7*	6.0	3.3**
Any drug[b]	29.0	13.7***	19.3	3.3***	24.1	8.4***
Any drug except marijuana[c]	14.7	8.1**	9.7	2.3***	12.2	5.1***

Note: Estimates are percentages. Ns (in parentheses) show the number of cases on which weighted data are based. Civilian data have been standardized to the military data by age, education, race/ethnicity, and sex.

*Different from civilian at the .05 level.

**Different from civilian at the .01 level.

***Different from civilian at the .001 level.

[a] Stimulants, sedatives, tranquilizers, and analgesics.

[b] Nonmedical use one or more times of marijuana or hashish, inhalants, hallucinogens, cocaine, heroin, or psychotherapeutics.

[c] Nonmedical use one or more times of drugs listed in footnote b except for marijuana or hashish.

Civilian data source: 1985 National Household Survey on Drug Abuse.

Military data source: 1985 Worldwide Survey.

greater drug use among the younger age group than among the older age group (about 10 percentage points for any drug use). This finding holds for both military and civilian populations. Thus, those ages 18 to 25 are at considerably higher risk [illegible] drug use than those who are older.

Cigarette Use

Military personnel are in general substantially more likely than civilians to be smokers and heavy smokers, as shown in Table 4. For the total group, the prevalence of smoking is about 5 percentage points higher among military personnel than among civilians, and the prevalence of heavy smoking is about 8 percentage points higher. Differences among military and civilian women are more pronounced than among military and civilian men. The rates of any smoking and heavy smoking among military and civilian men differ by 4 and 7 percentage points, respectively, while the same rates among military and civilian women differ by 13 and 14 percentages points, respectively. Military women are especially likely to smoke and smoke heavily: they have the highest prevalence of smoking (45 percent) and the same rate of heavy smoking as military men (30 percent). Rates of heavy smoking are particularly high among older military men and women, over one-third.

Discussion

After controlling for key sociodemographic characteristics known to be associated with substance abuse, clear differences remain in rates of substance use between military personnel and civilians. Military personnel are, in general, less likely to use drugs than civilians and more likely to drink and drink heavily and to smoke and smoke heavily. A striking finding is the extremely high rate of heavy drinking among younger military men (about one-third).

The substance use patterns of men and women are also of note. Among civilians, women are less likely than men to use drugs, alcohol, or cigarettes. Among military personnel, women are also less likely to use alcohol than men; in contrast, military women are equally as likely as military men to smoke and use drugs.

The precise factors accounting for differences in substance use are unknown but may plausibly be associated with distinctive conditions in military life and in military policies and programs directed toward substance use. The military policy of zero tolerance for drug use, which began in 1981 and is supported by urinalysis testing, has been a significant deterrent. Applicants to military service are tested for drug use, and active-duty personnel identified as users are subject to discharge from the military. This policy has, no doubt, been associated with rapid declines in drug use among military personnel during the 1980s, declines that have been much more rapid than those among civilians.

Further support for the deterrent effect of urinalysis testing comes from a recent study of military applicants.[12] Data from urine tests assessing the prevalence of marijuana and cocaine use found that applicants who knew they would be tested for drug use were less likely to test positive than those who did not know. These results suggest that some drug users who know about the screening program make sure they are "clean" before the test and that others decide not to apply. The decision of some drug users not to apply for military service probably accounts for some of the difference in military and civilian drug use.

TABLE 4—Standardized Comparisons of Cigarette Use in Past 30 Days Among Military Personnel and Civilians, by Age Group

	Age Group/Population					
	18–25 years		26–55 years		Total	
Group/Measure	Civilian	Military	Civilian	Military	Civilian	Military
Males	(N = 794)	(N = 1,632)	(N = 1,279)	(N = 5,857	(N = 2,073)	(N = 7,489)
Any smoking	36.9	43.0*	43.2	44.7	40.1	43.9
Heavy smoking	17.1	25.3***	28.4	34.8**	22.9	30.2***
Females	(N = 1,008)	(N = 208)	(N = 1,803)	(N = 340)	(N = 2,811)	(N = 548)
Any smoking	33.4	46.9*	30.5	42.7*	32.1	45.0***
Heavy smoking	15.7	26.2*	16.4	34.4***	16.0	30.0***
Total	(N = 1,802)	(N = 1,840)	(N = 3,082)	(N = 6,197)	(N = 4,884)	(N = 8,037)
Any smoking	36.5	43.4*	42.1	44.5	39.4	44.0*
Heavy smoking	17.0	25.4***	27.3	34.8***	22.2	30.1***

Note: Estimates are percentages. Ns (in parentheses) show the number of cases on which weighted data are based. Civilian data have been standardized to the military data by age, education, race/ethnicity, and sex.

*Different from civilian at the .05 level.

**Different from civilian at the .01 level.

***Different from civilian at the .001 level.

Civilian data source: 1985 National Household Survey on Drug Abuse.

Military data source: 1985 Worldwide Survey.

The higher rates of drug use among civilians suggest that military policies and practices effectively deter drug use in the military. In contrast, the lower rates of alcohol use and cigarette use among civilians than among military personnel suggest that certain aspects of military life may foster use and/or that military policies and programs directed toward alcohol and cigarette use have not been effective. Concerted efforts to decrease alcohol and cigarette use have been more recent and perhaps less intense than the efforts against drug abuse. The findings suggest the need for the military to intensify efforts to eliminate alcohol abuse and eradicate cigarette smoking. Military programs directed toward alcohol abuse should be targeted primarily toward young men who have the highest rates of heavy use. Programs against smoking should target both men and women.

Recent military policies and programs take a more stringent stance against alcohol abuse and smoking. These include an integrated health promotion program, an antismoking campaign, and specific policies to deglamorize and to limit accessibility to alcohol. This intensification of efforts promises further reductions in substance abuse. Data from the 1988 Worldwide Survey** show progress since 1985, with continuing significant reductions in the unstandardized rates of the prevalence of drug use (8.9 percent to 4.8 percent), alcohol use (86.6 percent to 82.8 percent), and cigarette use (46.2 percent to 40.9 percent) among military personnel in the past 30 days. Standardized comparisons conducted with the 1988 National Household Survey data, which will soon be available for public use, will reveal whether these recent deceases in use by military personnel and related decreases among civilians result in a continuing divergence of rates of use between military personnel and civilians or whether the rates are becoming more similar. Comparisons of the two sets of standardized comparisons will suggest whether the intensified military efforts against alcohol abuse and smoking have been effective. ☐

Acknowledgments

The research on which this article was based was funded by the Assistant Secretary of Defense (Health Affairs) under Contract No. MDA903-87-C-0763. The views, opinions, and findings contained in this paper are those of the authors and should not be construed as an official DoD position, policy, or decision, unless so designated by other official documentation.

References

1. Polich JM: Alcohol problems among civilian youth and military personnel. *In:* Blane HT, Chafetz ME (eds): Youth, Alcohol, and Social Policy. New York, NY: Plenum Press, 1979; 59–86.
2. Holcomb JF: Alcohol and the Armed Forces. Alcohol Health and Research World 1981/1982; Winter:2–17.
3. Bray RM, Marsden ME, Guess LL, Herbold JR: Prevalence, trends, and correlates of alcohol use, nonmedical drug use, and tobacco use among US military personnel. Milit Med 1989; 154:1–11.
4. Polich, 1979, p 69.
5. Polich JM: Epidemiology of alcohol abuse in the military and civilian populations. Am J Public Health 1981; 71:1125–1132.
6. Polich, 1979, pp 77–82.
7. Burt MR: Prevalence and consequences of alcohol use among US military personnel, 1980. J Stud Alcohol 1982; 43:1097–1107.
8. Allen J, Mazzuchi J: Alcohol and drug abuse among American military personnel: Prevalence and policy implications. Milit Med 1985; 150:250–255.
9. National Institute on Drug Abuse. National household survey on drug abuse: Main findings 1985. Rockville, MD: National Institute on Drug Abuse, 1988.
10. Little RJA: Direct standardization: A tool for teaching linear models for unbalanced data. Am Stat 1982; 36:38–43.
11. Mulford HA, Miller DE: Drinking in Iowa II. The extent of drinking and selected sociocultural categories. J Stud Alcohol 1960; 1:26–39.
12. Vogl WF, Peterson MR, Jewell JS: Prevalence of drug use among applicants for military service—United States. June–December 1988. MMWR August 25, 1989, 38:580–583.

**Bray RM, Marsden ME, Guess LL, Wheeless SC, Iannacchione VG, Keesling SR: 1988 Worldwide survey of substance abuse and health behaviors among military personnel. Report prepared for the Assistant Secretary of Defense (Health Affairs) by Research Triangle Institute, Research Triangle Park, NC, 1988.

Risk Factors for Suicide Attempts Among Navajo Adolescents

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ABSTRACT

Background: Rates of adolescent suicide in the United States are highest among Native Americans but little is known about risk factors for suicide attempts in this population.

Methods: To identify risk factors for self-reported suicide attempts by Navajo adolescents, we analyzed the 1988 Indian Health Service Adolescent Health Survey that was administered to 7,254 students in grades 6 through 12 on the Navajo reservation. The responses of students reporting a past suicide attempt were compared to others.

Results: Nearly 15 percent (N = 971) reported a previous suicide attempt; over half of those admitted to more than one attempt. Controlling for age, a logistic regression model revealed the following associations with suicide attempts: a history of mental health problems (OR = 3.2); alienation from family and community (OR = 3.2); having a friend who attempted suicide (OR = 2.8); weekly consumption of hard liquor (OR = 2.7); a family history of a suicide or attempt (OR = 2.3); poor self-perception of health (OR = 2.2); a history of physical abuse (OR = 1.9); female gender (OR = 1.7); and sexual abuse (OR = 1.5).

Conclusions: Efforts to prevent adolescent suicide attempts in this population should target individuals with those risk factors of the highest risk and prevalence of exposure. (*Am J Public Health*. 1991;81:870–874)

Introduction

In 1987, over 2,100 youths under age 19 in the United States killed themselves, making suicide the third most common cause of death in the US adolescent population. Although suicide rates in the US population have been relatively stable, rates among youths have tripled over the past 30 years, from 4.5 per 100,000 in 1950 in 13.1 per 100,000 in 1986.[1] Among US ethnic groups, American Indians and Alaskan Natives have the highest rates.[2,3] The suicide rate among Native American adolescents in 1987 was 26.8 per 100,000, more than double the national rate for adolescents of all races.[4]

For every completed suicide, it is estimated that eight or more are attempted.[5] The 1987 National Adolescent Student Health Survey reported that 11.1 percent of males and 17.5 percent of females from a national random sample of eighth and tenth grade students had harmed themselves in a way that might have caused their death.[6] The prevalence of suicide attempts among Native Americans may even be higher. Among a cohort of American Indian high school students attending a boarding school in the southeast US, 23 percent had attempted suicide during their lifetime.[7]

This high rate of self-destructive behavior among Native American adolescents warrants further investigation and culturally appropriate intervention. The 1988 Navajo Adolescent Health Survey offered an opportunity to examine a set of traditional risk factors that are thought to be important in the general population. This population was chosen because it had the largest and most complete participation in the Indian Health Service Adolescent Health Survey conducted in 1988. The purpose of this study was to identify risk factors for self-reported suicide attempts among adolescent students in the Navajo Indian population.

Methods

Instrument

The source of data was the 1988 Navajo Adolescent Health Survey, a subset of the Indian Health Service Adolescent Health Survey. This 162-item survey was designed as a health promotion tool for use by the Indian Health Service and individual communities to identify and address problems in adolescent health. The original instrument was designed by Adolescent Health Program of the University of Minnesota[8] and adapted for use in Native American populations. After pilot testing, the survey was administered to Native American students throughout the United States. Content of the survey included personal data, information about the home and family, general health, nutrition, substance abuse, social concerns, experiences and attitudes regarding sex, mental health, and injury. The data from the Navajo Reservation represented more than 70 percent of the national sample.

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TABLE 1—Crude and Adjusted Odds Ratios of Risk Factors for Suicide Attempts

	Odds Ratios		
Category/Risk Factor	Crude	Adjusted*	95% CI
Child Abuse			
Physical abuse	4.0	1.9	1.5–2.4
Sexual abuse	3.4	1.5	1.2–1.9
Demography			
Female gender	1.8	1.7	1.4–2.0
Community of residence	1.4 (Community "D")	NS	
Exposure to Suicide			
Family history of attempt/completion	3.9	2.3	1.6–3.2
Friend history of attempt	5.0	2.8	2.3–3.4
Friend history of completion	3.7	NS	
Family Structure			
Biological mother absent	.8	NS	
Biological father absent	1.3	NS	
Parents separated	1.5	NS	
One parent dead	1.4	NS	
Self-perception of Health Status			
Poor Health	4.4	2.2	1.3–3.8
Mental Health			
History of mental, behavior problems requiring professional help	3.7	3.2	2.2-4.5
Extreme alienation from family & community	5.0	3.2	2.1–4.4
Substance Abuse			
Weekly use of beer/wine	1.4	NS	
Weekly use of hard liquor	4.2	2.7	1.9–3.9
Mother's daily use of beer/wine	2.6	**	
Mother's daily use of hard liquor	4.3	**	
Father's daily use of beer/wine	2.6	**	
Father's daily use of hard liquor	3.6	**	
School Perception and Performance			
"I hate School"	2.3	NS	
Less Than "C" Grade Point Average	2.0	**	

NS: Not significant after adjustment
*Adjusted for age & all covariates
**"N" Too small to enter regression (see text)

Study Site/Subjects

The Navajo Reservation is the largest Indian nation outside of Alaska with an estimated population of 190,000. Schools on or adjacent to the reservation were encouraged to participate, although each school or individual student could decline. A volunteer sample of 47 schools representing grades six to twelve participated in the survey. All students in participating schools were asked to voluntarily respond to the anonymous survey during class time. There was no attempt to survey students absent on that day. The response rate exceeded 70 percent of enrolled students in virtually all participating schools to yield a usable sample of 7,241 responses, representing approximately one-third of all eligible students on the Navajo reservation.

Risk Factors

To determine risk factors for self-report of attempted suicide, we analyzed this survey using one question as the outcome, or dependent, variable: "Have you ever tried to kill yourself?" The independent variables consisted of 28 survey items in eight categories (Table 1) hypothesized to be underlying risk factors for suicide attempts. There were one to six survey items in each category. All of the items, except alienation, were measured by a single survey question.

Alienation was assessed using a nine item scale (Table 2) that explores the respondent's connectedness with the family and community. This scale had substantial internal consistency (Cronbach's alpha = 0.85) and construct validity when compared to other items of a similar nature in the survey.

Students reporting a suicide attempt were compared to those with no such report for each of the hypothesized risk factors. Chi-square statistics were used to establish significance in these comparisons and the significance level was adjusted to .0018 by the Bonferroni correction for multiple comparisons. Items that met this level of significance were entered in a multiple regression equation controlled for age and gender. Both forward stepwise and structured regression strategies were employed in this procedure. Using F tests, items that remained significant after adjustment for the other variables were entered into a logistic model. Estimates of adjusted odds ratios for each risk factor were derived from the coefficients of the logistic regression model. Categorical variables were divided into a series of dichotomous dummy variables; odds ratios presented for these variables represent comparison of the high versus low exposures. Data were analyzed with the SPSS-PC (SPSS Inc., Chicago, IL 60611) and EGRET (Statistics and Epidemiology Research Corp., Seattle, WA 98105) statistical packages.

Results

The sample was 51 percent female with a median age of 14.4 years and ninth

TABLE 2—Alienation Scale Items

How much do you feel:

1. Adults care about you?
2. School people care about you?
3. Your parents care about you?
4. Church leaders care about you?
5. Tribal leaders care about you?
6. Your family cares about your feelings?
7. People in your family understand you?
8. You and your family have lots of fun together?
9. Your family pays lots of attention to you?

5 point Likert Scale (Not at all—Very much)
1 2 3 4 5
Reliability (Cronbach's alpha) coefficient = 0.85
Mean score: 32.3 (SD 8.0)

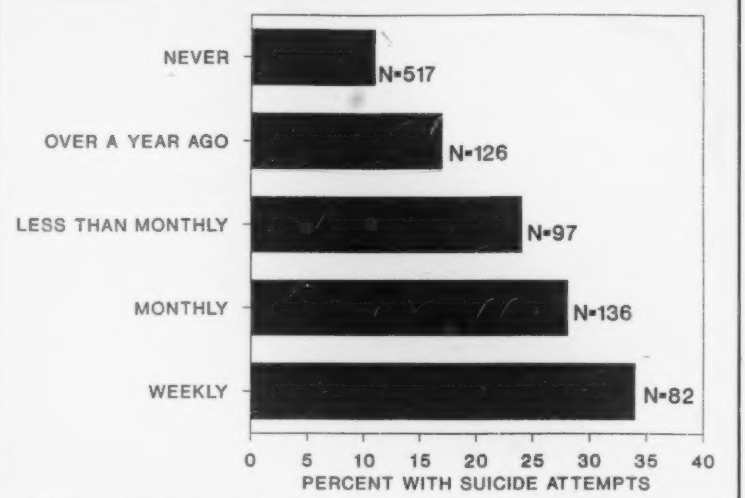

FIGURE 1—Suicide Attempts by Hard Liquor Use among Navajo Adolescents.

grade standing. Exposure to suicidal behavior among friends and family was common in this group of students. About 18 percent of the respondents replied that someone in their family had tried to kill or had killed themselves. Similarly, almost 18 percent had a friend who had attempted suicide and 9 percent had a friend who had completed suicide.

Of all respondents, 6,637 (92 percent) answered the question about suicide attempts; 971 (15 percent) admitted to a past attempt to kill themselves. Thirty-five percent of these attempts had occurred more than one year before the survey and 58 percent of the students had tried to kill themselves more than once. Thirty-six percent of suicide attempts were not reported to anyone and only 15 percent were brought to the attention of a medical professional.

In Table 1, the crude and adjusted odd ratios for hypothesized risk factors that were significant in distinguishing between respondents who had reported a previous suicide attempt, and those who had not, are shown.

In the final model, nine risk factors demonstrated a statistically significant and independent association with a history of suicide attempts. The two strongest associations were a history of a mental, behavioral, or emotional problem requiring professional help (OR = 3.2) and extreme alienation from family and community (OR = 3.2). Exposure to suicide completions and attempts by family and friends also shared strong associations with suicide attempts. Having a friend who previously attempted suicide (OR = 2.8) was more strongly associated with attempts by the respondent than having a family member either attempt or complete suicide (OR = 2.3). The consumption of hard liquor at weekly intervals was also significantly associated (OR = 2.7) with suicide attempts. The frequency of hard liquor consumption was linearly correlated with a lifetime prevalence of suicide attempts (Figure 1). Beer and wine consumption were not significant correlates at any frequency of use after adjustment for hard liquor use.

A self-perception of poor general health was also independently associated with a history of suicide attempts (OR = 2.2). The association of past physical abuse (OR = 1.9) with suicide attempts was marginally stronger than that of sexual abuse (OR = 1.5). Finally, the odds ratio for female gender remained significant after adjustment for covariates (OR = 1.7).

Several risk factors appeared to be associated with a past suicide attempt in the bivariate analysis but were not included in the model because of the many missing values. These variables included both the maternal and paternal consumption patterns of alcohol and the respondent's school performance as measured by the grade point average.

Risk factors which were significant in the bivariate analysis but did not remain significant after adjustment for other covariates, or after correction for multiple comparisons, included: community of residence, history of friend's suicide completion, all of the family structure variables, the weekly use of beer or wine, household density, religiosity of the respondent, the household presence of the natural mother, and an adoptive father or stepfather.

Discussion

To our knowledge, this is the first large controlled study of suicide attempts among Native American adolescents. The lifetime prevalence of self-reported attempts among Navajo students is high, but similar to that reported in the Minnesota and National Adolescent surveys.[6,8] The risk factors ascertained in this study of Native American youth are similar to those described for the general population.

An important finding of this study is the link between the suicide attempts of the respondents and those of their friends and family. Because prevalence of exposure was the highest for these risk factors and because of their strong association with suicide attempts, these risk factors have the greatest public health importance. Although bereavement following a family suicide is recognized to be a risk factor for suicidal behavior in older adults,[9] there have been few controlled studies documenting this risk in children and adolescents. The studies of adolescent suicide clusters, where a number of suicide deaths occur in a community over a short period, have been conflicting as to whether exposure to a friend's suicide is a risk factor.[10–12] But these studies have only evaluated deaths, not attempts, and have studied only suicide deaths associated with a suicide cluster.

Alienation from family and community was independently associated with a history of previous suicide attempt. This supports findings from numerous uncontrolled studies in other populations, including Durkheim's sociologic analysis of suicide.[13]

The association of alcohol and suicide attempts confirms previous findings and demonstrates new ones. Chronic and acute ethanol intoxication appear to play an important role in completed suicides of both White and Native Americans.[14,15] In contrast, there are few data on the role of alcohol in suicide attempts, nor on the differences between varying types of alcohol. After adjustment for beer and wine consumption, use of hard liquor remained the significant alcohol-associated variable related to suicide attempts. The linear relationship between frequency of hard liquor use and the lifetime prevalence of suicide attempts suggests that its consumption (or a close proxy) may play a causal role in suicide attempts.

The finding that past sexual or physical abuse is a risk factor for suicide attempts correlates with reports from other populations. Riggs, *et al*, recently reported similar findings for sexual and physical abuse in a smaller survey of Rhode Island students, although these variables were not adjusted for other behavior and life events.[16] Our study also indicated an interaction between the gender and abuse variables, demonstrating that sexual abuse of males and physical abuse of females are associated with higher risks than the converse situations. This has not been described in other studies.

Mental health problems were expected to be risk factors as their association to suicidal behavior has been well described.[17–19] Despite this, few controlled studies have confirmed this finding in the American Indian population. Manson's survey of Indian adolescent students from a southeastern boarding school demonstrated that those with a past suicide attempt had a higher level of depressive symptomatology.[7] A recent controlled study of adolescents presenting to an urban emergency room for suicide attempts indicated that they were more likely than controls to have a history of mental health care.[20]

There are several limitations to this study. Its cross-sectional design did not allow us to establish the temporal relationship between these risk factors and the suicide attempt. Mental health problems or alienation, may show associations because they may also be sequelae of an attempt. In addition, some associations may represent proxies for other variables not in the model. An example of a potential proxy might be child abuse, where the abuse history is important because it is symbolic of pervasive family dysfunction.

The survey did not measure the severity of the self-inflicted injury. Since only 15 percent of the attempts were reported to medical professionals, we infer that the unreported attempts represented gestures or minor injuries. Alternate interpretations are that potentially lethal attempts were only marginally successful, or that some of these unreported attempts were disguised as unintentional injuries, e.g. single occupant motor vehicle crash.

Because the study was conducted in school, it did not include those students absent on the day of the survey. Students who had been expelled, suspended, or had dropped out before the survey also were not represented. All of these groups may be at higher risk for many of these behaviors and life events. This may have led to an underestimate of the true lifetime prevalence of suicide attempts.

Although the use of anonymous self-reported questionnaires precluded corroboration of responses with existing data sources such as medical and social service records, its anonymity may have been an important safeguard of its validity. Since many of these behaviors are illicit or taboo, adolescents may be reluctant to disclose such information in a survey with identifiers. The validity of the self-reported information is supported by the similarity of these results to those from other studies.

Finally, this survey did not include some potentially important risk factors in this population. Exposure to traumatic deaths other than suicides and adaptability to a bi-cultural environment were not included but may also be important risk factors.[21,22]

The causal model of suicidal behavior is extremely complex and appears to vary between age and cultural groups. The causal model will be constructed using risk factors derived from controlled studies. Future preventive strategies should focus on identified risk factors that can be modified.[23] Furthermore, public health efforts to reduce suicide rates will be most successful if focused on those risk factors with the highest attributable risk. Future research efforts to determine the etiology of suicidal behavior in the American Indian and Alaskan Native populations should be directed toward carefully designed population-based case-control studies. ☐

Acknowledgments

We thank Frederick Rivara, MD, and Robert Putsch, MD, for their helpful comments on the manuscript and Silvana Chatrian for her assistance. This work was completed while Dr. Grossman was a Robert Wood Johnson Clinical Scholar. Also supported in part by a grant from the Indian Health Service and by the Northwest Health Services Research and Development Field Program, and the Seattle Veterans Affairs Medical Center. The opinions, conclusions, and proposals expressed in this article are those solely of the authors and do not necessarily represent the views of the Robert Wood Johnson Foundation or the Indian Health Service. Presented, in part, at the Ambulatory Pediatric Association, Anaheim, CA, May 1990.

References

1. Youth Suicide Surveillance, Centers for Disease Control, Atlanta, GA, 1986.
2. Berlin IN: Prevention of adolescent suicide attempts among Native American tribes. Adolesc Psychiatry 1985;12:77–93.
3. Kraus RF, Buffler PA: Sociocultural stress and the American Native in Alaska: An analysis of changing patterns of psychiatric illness and alcohol abuse among Alaska Natives. Culture, Med Psychiatry 1979;3:111–151.
4. Chart-Series Book, Indian Health Service, US Public Health Service 1988; 43.
5. Rosenberg ML, Smith JC, Davidson LE, Conn JM: The emergence of youth suicide: An epidemiologic analysis and public health perspective. Annu Rev Public Health 1987;8:417–440.
6. American School Health Association, *et al*: The National Adolescent Student Health Survey, A Report on the Health of America's Youth. Oakland, CA: Third Party Publishing, 1989.
7. Manson SM, Beals J, Dick RW, Duclos C: Risk factors for suicide among Indian adolescents at a boarding school. Public Health Rep 1989;104:609–614.
8. Blum R, Resnick M, Geer L, *et al*: The Minnesota Adolescent Health Survey: Implication for physicians. Minnesota Med 1988;71:143–146.
9. Monk M: Epidemiology of Suicide. Epidemiol Rev 1987;9:51–69.
10. Davidson LE, Rosenberg ML, Mercy JA, Franklin J, Simmons JT: An epidemiologic study of risk factors in two teenage suicide clusters. JAMA 1989;262:2687–2692.
11. Gould MS: Teenage suicide clusters (letter). JAMA 1990;263:2051–2052.
12. Gould MS, Wallenstein S, Kleinman M: Time space clustering of teenage suicide. Am J Epidemiol 1990;131:71–78.
13. Durkheim E: Suicide. New York: The Free Press, 1951.
14. Brent DA, Perper JA, Allman CJ: Alcohol, firearms, and suicide among youth. JAMA 1987;257:3369–3372.
15. Hlady WG, Middaugh JP: Suicides in Alaska: Firearms and alcohol. Am J Public Health 1988;78:179–180.

16. Riggs S, Alario AJ, McHorney C: Health risk behaviors and attempted suicide in adolescents who report prior maltreatment. J Pediatr 1990;116:815–821.
17. Brent DA, Perper JA, Goldstein CE, Kolko DJ, Allan MJ, Allman, CJ, *et al*: Risk factors for adolescent suicide. Arch Gen Psychiatry 1988;45:581–588.
18. Shaffer D: The epidemiology of teen suicide: An examination of risk factors. J Clin Psychiatry 1988;49 (9 suppl):36–41.
19. Joffe RT, Offord DR, Boyle MH: Ontario Child Health Study: Suicidal behavior in youth age 12–16 years. Am J Psychiatry 1988;145:1420–1423.
20. Slap GB, Vorters DF, Chaudhuri S, Centor RM: Risk factors for attempted suicide during adolescence. Pediatrics 1989;84:762–772.
21. Rubinstein D: Epidemic suicide among Micronesian adolescents. Soc Sci Med 1983; 17:657–665.
22. Levy JE, Kunitz SJ: A suicide prevention program for Hopi youth. Soc Sci Med 1987;25:931–940.
23. Shaffer D, Garland A, Gould M, Fisher P, Trautman P: Preventing teenage suicide: A critical review. J Am Acad Child Adolesc Psychiatry 1988;27:675–687.

Job Stress Is "Epidemic," Costly to Business, Study Finds

One in three Americans seriously thought about quitting work in 1990 because of job stress, and one in three expects to "burn out" on the job in the near future.

A recent study also claims that workplace stress is causing far greater turnover, lower productivity, higher absenteeism, and more frequent health problems than was previously recognized. Employees who are overworked or have little control over their job are most likely to be burnout victims.

These are among findings from a study prepared by Northwestern National Life (NWNL) Insurance Company identifying the extent, causes, costs, and solutions related to workplace stress. Unlike many earlier studies, the NWNL research is believed to be the first to correlate causes and cures in all occupations and populations across the United States.

Any major change in the workplace affecting staffing is likely to speed up employee burnout, the research suggests. Significant burnout occurred in companies that had substantially cut employee benefits, changed ownership, required frequent overtime, or reduced the work force.

The findings are from interviews with a representative sample of 600 American workers. To help address the problem, NWNL also assembled a panel of stress and wellness experts to construct a workplace stress test that enables a company to quickly measure its work force stress and identify the consequences and solutions.

Among NWNL's key findings are the following:

- Seven in 10 American workers say that job stress is causing frequent health problems, and is making them less productive.
- Thirty-four percent of Americans thought seriously of quitting their job in 1990 because of workplace stress. Fourteen percent of all employees said workplace stress actually caused them to quit or change jobs in the last 2 years.
- Forty-six percent say their job is highly stressful. This is double the findings of a 1985 National Health Interview Survey that said 20% of Americans experience a high level of stress.
- Seventeen percent report higher absenteeism due to job stress.
- Fifty-three percent of Americans are required to work more than 40 hours a week very or somewhat often.
- Eighty-two percent of workers say victims of burnout deserve disability pay from their employer.
- Peggy Lawless, NWNL's research analyst, noted that job stress and burnout have a foothold in the US work force without regard to region, size of company, type of job, or age of the worker.

Lawless said the survey also shows that companies with supportive work and family policies, health coverage for mental illness and chemical dependency treatment, improved communication, and flexible work hours have almost half the burnout rate of companies that don't have such policies.

Lawless likened workplace stress in the 1990s to the lack of workplace safety in the 1960s. "Stress is less visible than coal dust or dangerous machines, but it's more prevalent and just as damaging to health and productivity."

The research results and the NWNL Workplace Stress Test are contained in a copyrighted report by NWNL called "Employee Burnout: America's Newest Epidemic." Copies can be obtained by calling 612/342-7137.

Patterns of Mental Health Utilization Among Island Puerto Rican Poor

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ABSTRACT

Background: This paper describes utilization of mental health services by poor Puerto Ricans living on the island. It examines the utilization rates, within health sectors, and settings for the provision of mental health services.

Methods: Data are based on an islandwide probability sample of 18- to 64-year-old respondents living in low socioeconomic areas. We assessed need with the Psychiatric Symptom and Dysfunction Scales.

Results: Approximately one-third of our study population (31.5%) met criteria for need. Of these, only 32% had received any mental health care in the past year. Need was significantly associated with use of physical or mental health services for mental health problems. We found those who needed services to be five times more likely than those who did not need services to have used one or both sectors of care at least once in the past year. Among the first group 21.8% used the physical health sector to deal with mental health problems in contrast with 17.9% who sought care in the mental health sector. In the physical health sector, subjects used the public and private settings equally. In the mental health sector, 70% of subjects used the public setting.

Conclusions: This suggests the nonpsychiatric physician as a main provider for mental health treatment. (*Am J Public Health*. 1991;81:875–879)

Introduction

We report the distribution of mental health services provided by the medical (physical) and mental health sectors to low income people living in Puerto Rico. The relation of sociodemographic characteristics with utilization of mental health services is examined. Utilization data obtained in this study provide information on the gap between the need for and provision of mental health care.

Researchers have studied the use of mental health and health services by various ethnic minorities.[1–7] Some studies have found underutilization of mental health services by Hispanics,[2–5] while others question this underutilization.[6–7] Discrepant findings have been attributed to treatment surveys versus community surveys, to the criteria used to define Hispanics, and to differences in the need for mental health services.

Previous studies[8–10] indicate that persons of lower socioeconomic status (SES) have a low rate of mental health service utilization. A recurring argument is that the poor hold attitudes and ideologies dissonant with scientific medicine.[11,12] Hoppe and Heller[13] suggest that lower-class populations distrust those outside of their circle of family and friends, having heavy involvement in familial and peer relationships. Differences in values and priorities placed on health across socioeconomic groups may better explain observed differences in the use of mental health and physical health sectors. For example, Ware and Young[14] reported that persons of lower SES value their physical health more than their mental health. Johnston and Ware[15] report that SES appears associated with somatization, recognition of illness symptoms, and behaviors in response to illness. Consistent evidence has shown that a substantial proportion of persons with emotional problems and mental disorders are treated in the physical health sector.[16–20] Hough, *et al*,[21] found that although Mexican Americans made fewer visits to mental health providers than their non-Hispanic counterparts, they did not differ in the proportion of visits to a physical health provider for mental health reasons.

This study concentrates on the population in the lowest socioeconomic strata since there is evidence to suggest this group is greatly exposed to economic strains, physical illness, and disabilities, known risk factors for mental health needs.[22,23] Little is known to explain the use of mental health services for individuals that belong to the lowest income strata.[24] In this paper, we examine the utilization of mental health services by this population on the island of Puerto Rico.

Methods

The target population of this study included the civilian, noninstitutionalized residents, ages 18 to 64, living in low so-

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cioeconomic areas of Puerto Rico. The population was sampled using a two-stage stratified cluster sample of the island. The strata were urban and rural, which economically defined sub-strata. The substrata or segments were classified as economically depressed based on an index of media house rent, family income, and house value. These segments were divided into clusters from which 178 were selected for our sample. The list of clusters defined a survey area which contained about 57 percent of the total island population living in poverty. The nonpoor living in these areas were surveyed but excluded from the analyses reported here. The poor people living in nonpoor areas were also excluded from the sample. The sample for this study was obtained using the sampling frame prepared by the Department of Labor and Human Resources, and based on the 1980 Census of Population and Housing as updated by new housing developments in 1986.

The probability sample of clusters yielded 2,682 housing units. Of these, 27.9 percent (748 housing units) were not eligible. Reasons for ineligibility included vacancies (292) and noneligible residents (456) 18 to 64 years of age.

One respondent in each household was randomly selected from among those eligible. The noninterview rate was 8.1 percent distributed as: 2.5 percent refusals; 5.4 percent persons unable to be contacted after three callbacks; and 0.3 percent other breakoff. A total of 1,777 respondents were successfully interviewed during February to October 1989, yielding a completion rate of 91.9 percent. We constructed a poverty indicator based on size of family and number of related children according to the criteria of the Department of Commerce, Bureau of the Census.[25] Of those respondents living in poor areas, 73.6 percent were assessed to be in poverty status according to the 1989 Census definition. Our analyses are limited to this subpopulation.

All analyses incorporate the underlying survey design. Estimation and testing was performed using the program PC Carp.[26] Group differences in sociodemographic characteristics for utilizers of the physical health sector and the mental health sector for mental health services were tested using Z scores without multiple comparison adjustments. Sociodemographic differences with regard to service sectors used were tested with analyses of variance. Differences are not discussed unless the p value was less than 0.05. In all comparisons we will discuss the weighted sample so our inferences will apply to the Puerto Rican poor population residing in low socioeconomic status areas.

Measures

Respondents were interviewed by lay interviewers who gathered data on sociodemographics, psychiatric symptomatology, physical health, and utilization of physical health and mental health services. Questions on utilization of physical and mental health services for treatment of mental health problems were based on those of the National Institute of Mental Health (NIMH) Epidemiologic Catchment Area studies.[20] For the purposes of this paper, health care use will be limited to the formal system of care, recognizing that the informal system of care (e.g. clergy, friends, family) may also be an important link for explaining how Puerto Ricans cope with their emotional problems.

A mental health care visit is defined as a contact with any type of formal health care provider for a mental health problem. Included in the definition are visits to any type of formal health facility, such as a community health center, or to a physical health provider to deal with mental health problems. The mental health related contacts are examined in the physical health sector and the mental health sector. The physical health sector represents visits to a non-psychiatric physician for mental health problems. The mental health sector corresponds to visits to psychiatrists, psychologists, or any mental health treatment program regardless of whether there was a mental health reason for the visit.

Psychiatric symptomatology was assessed by the Psychiatric Symptom and Dysfunction Scales (PSDS). It consists of five subscales: anxiety, depression, psychosocial dysfunction, cognitive impairment, and general psychopathology. The items for these subscales were selected from the psychiatric, epidemiologic, and clinical research literature for the Florida Health Study.[27-30] Psychiatric symptoms and nondiagnosed psychiatric disorder were the criteria employed to determine need for mental health services. The scale provides a normative distribution of psychiatric symptomatology and dysfunctions in the population. They do not diagnose individuals. The PSDS has been found to be a valuable and adequate measure to identify differential levels of mental health need within the general and patient populations, including Hispanics and other ethnic groups on the mainland.[28,29] Persons scoring with moderate or high symptoms on 3 to 5 scales are included in the need category[2] for mental health services and are termed "needers."

These scales were adjusted for number of chronic illnesses, since some PSDS scores increased with the number of chronic illnesses. Specifically, psychosocial dysfunction, anxiety, and depression scores were reduced two, two, and three points, respectively, for every chronic health illness reported by the respondent. Cognitive impairment and general psychopathology scores were reduced by one point for each chronic physical illness. The actual construction of the scale permits no probing to differentiate whether the apparent psychiatric symptom (e.g. sweaty hands) has some physiological origin. Therefore, adjusting the scales in this respect reduces the upward bias due to poor physical health.

Results

The sociodemographic characteristics of respondents who utilized the mental or physical health sectors for mental health reasons are presented in Table 1. In general, utilization of physical health services did not vary with age. Use of mental health services was significantly higher for those 45–54 years old ($p < .05$) compared to those 18–24 and 35–44 years of age. A higher percentage of respondents with less than 10 years of school education than those with higher scholastic achievement reported use of physical health services for mental health reasons during the last year. Use of mental health services was significantly lower among respondents with some college education ($p < .01$) in comparison to those with a lower educational level. The retired or incapacitated were more likely than others to use the mental health sector or both sectors to deal with emotional problems. Sex, marital status, insurance, and zone of residence did not show a statistically significant association with health services sectors used for mental health reasons.

The utilization of the health and mental health care sector for mental health reasons during the previous year is shown in Table 2. As expected, needers are five times as likely to use one or both sectors of care at least once in the past twelve months for mental health problems (31.5 vs 5.8 percent). This discrepancy is even greater when we consider the use of the general health sector at least on one occasion (21.8 vs 3.7 percent) during the last year. These visits were identified by the respondents as health encounters to deal with mental health problems. The utilization of the mental health sector showed a higher prevalence of mental health visits by needers than by non-needers (17.9 vs 2.3 percent). Among users, the intensity varied by sector and need status. Needers averaged 7.3 (±0.9) visits to the physical health providers while non-needers averaged only 3.5 (±0.6) visits ($p < 0.001$). In contrast, there were no significant differences in the mental health sector utilization visits by needers (9.6, ± 1) versus 11.1 ± 2.7 visits by non-needers ($p > 0.25$).

Nearly 13.6 percent of needers of mental health services are receiving care exclusively in the physical health sector (row 4, Table 2). Furthermore, there is a small group of needers who use both sectors for mental health reasons (8.2 percent). This dual usage of mental health services in both the physical and mental health sector rarely occurs among non-needers (0.3 percent).

Table 3 presents the utilization during the last year of the public and private settings for a mental health problem by need status. Utilizers of exclusively the physical health sector were equally likely to choose a private or public setting for mental health care. In contrast, utilizers of exclusively the mental health sector employed by a 3 to 1 margin the public over the private setting. Approximately one-half of the users of public mental health services reported using mental health centers (data not shown).

TABLE 1—Sociodemographic Characteristics of the Sample Below Poverty Level by Health Sector Utilized for Mental Health Reasons During the Last Year

	Utilizers of Services for Mental Health Problems			
Sectors:	Physical Health	Mental Health	Both	Non-users
Population N	46,980	31,590	19,710	609,390
Sample n	96	59	42	1,111
	% (S.E.)	% (S.E.)	% (S.E.)	% (S.E.)
Age (years)				
18–24	16.7 (5.0)	12.0 (3.9)	5.5 (3.3)	25.9 (1.5)
25–34	17.2 (3.6)	21.4 (5.2)	12.3 (4.4)	26.2 (1.5)
35–44	21.3 (4.7)	16.2 (5.0)	24.7 (7.7)	21.0 (1.4)
45–54	25.9 (5.3)	32.5 (7.4)	28.8 (8.2)	13.2 (1.2)
55–64	19.0 (4.0)	17.9 (5.7)	28.8 (6.9)	13.6 (1.2)
Education (years)				
0–6	33.9 (5.6)	35.9 (7.6)	41.1 (9.3)	24.7 (1.4)
7–9	26.4 (6.0)	15.4 (5.0)	23.3 (7.1)	20.4 (1.2)
10–11	10.9 (3.3)	18.8 (5.8)	2.7 (1.9)	9.9 (1.0)
12	16.7 (4.3)	25.6 (6.4)	17.8 (6.2)	28.0 (1.4)
13+	12.1 (4.3)	4.3 (2.4)	15.1 (5.8)	17.0 (1.3)
Sex				
Male	33.9 (5.5)	41.9 (1.1)	57.5 (1.2)	39.8 (1.5)
Female	66.1 (5.5)	58.1 (7.6)	42.5 (8.4)	60.2 (1.5)
Marital Status				
Not Married	43.1 (5.8)	46.2 (6.8)	37.0 (7.6)	40.4 (1.8)
Married	56.9 (5.8)	53.8 (9.6)	63.0 (1.1)	59.6 (1.8)
Employment				
Employed	30.1 (6.1)	11.5 (4.3)	12.5 (7.1)	40.7 (2.0)
Unemployed	39.1 (7.0)	22.1 (5.9)	9.4 (3.7)	34.1 (1.9)
Retired or Incapacitated	21.1 (4.9)	56.7 (8.1)	71.9 (8.2)	10.0 (1.1)
Student	9.8 (5.7)	9.6 (3.9)	6.3 (4.0)	15.3 (1.5)
Insurance				
No	85.6 (3.9)	84.6 (7.3)	69.9 (1.1)	87.6 (1.3)
Yes	14.4 (3.9)	15.4 (5.2)	30.1 (7.9)	12.4 (1.3)
Zone of Residence				
Urban	46.6 (5.6)	57.3 (7.0)	53.4 (9.1)	55.7 (1.9)
Rural	53.4 (5.6)	42.7 (10.0)	46.6 (1.3)	44.3 (1.9)

TABLE 2—Utilization in the Past Year of Physical Health and Mental Health Services for Mental Health Reasons

		Total Sample	Needers	Non-needers
Respondents with one or more visits to:	Population N Sample n	707,670 1,308	222,750 413	484,920 895
		% (S.E.)	% (S.E.)	% (S.E.)
Any Sector		13.9 (1.1)	31.5 (2.6)	5.8 (0.8)
Physical Sector⁺		9.4 (0.9)	21.8 (2.4)	3.7 (0.6)
Mental Health Sector⁺		7.3 (0.8)	17.9 (1.9)	2.3 (0.5)
Only the Physical Health Sector		6.6 (0.7)	13.6 (2.0)	3.5 (0.6)
Both Physical Health and Mental Health Sector		2.8 (0.5)	8.2 (1.5)	0.3 (0.2)

Numbers in parentheses indicate standard errors.
⁺Does not exclude the use of other sectors.

Discussion

In contrast to other studies,[18,31,32] poor women in our sample were as likely to receive mental health care as men; elevated rates of unemployment (38 percent) among the poor may be a contributing factor to this finding. Unemployment also may be related to diminished social standing and vulnerability to psychological distress in men, leading them to seek mental health services.

Insurance did not appear to facilitate entrance into mental health care. This could be an artifact of Medicaid eligibility, since it was reported by our respondents as noninsurance. Medicaid operates as a centrally administered program by the Department of Health, whereby participants have free access to public health facilities. Public health and mental health services have an open door policy which may render those services accessible to the "uninsured."

Total mental health visits among poor Puerto Ricans (31.5 percent in our study sample) were twice as high as those reported in the ECA sites.[20] Economic factors and impoverished social conditions may contribute to a higher need for mental health services.

The finding that almost 22 percent of the needers went to professionals who are not specialists in mental health suggests a possible link between mental health disorders and physical illness, or difficulties in discriminating psychiatric symptoms from somatic complaints as a cultural expression of discomfort. Findings by

TABLE 3—Utilization of the Physical Health and Mental Health Sectors by Setting and Need Status (previous year) for the Sample Below Poverty Level

Sector		Total	Needers	Non-needers
	Population N	98,280	70,200	28,080
	Sample n	197	137	60
All Sectors		% (S.E.)	% (S.E.)	% (S.E.)
Private setting*		51.1 (3.9)	52.3 (4.7)	48.1 (7.5)
Public setting		48.9 (3.9)	47.7 (4.7)	51.9 (7.5)
	Population N	46,980	30,240	16,740
	Sample n	96	59	37
Physical Health All Sectors		% (S.E.)	% (S.E.)	% (S.E.)
Private*		55.7 (5.8)	53.6 (7.5)	59.7 (8.9)
Public		44.3 (5.8)	46.4 (7.5)	40.3 (8.9)
	Population N	31,590	21,600	9,990
	Sample n	59	40	19
Mental Health		% (S.E.)	% (S.E.)	% (S.E.)
Private setting*		30.8 (6.5)	32.5 (8.0)	27.0 (10.5)
Public setting		69.2 (6.5)	67.5 (8.0)	73.0 (10.5)
	Population N	19,710	18,360	1,350
	Sample n	42	38	4
Both Physical and Mental Health		% (S.E.)	% (S.E.)	% (S.E.)
Private*		72.6 (7.8)	73.5 (8.0)	60.0 (24.0)
Public		27.4 (7.8)	26.5 (8.0)	40.0 (24.0)

Note: Numbers in parentheses indicate standard errors.
*Private includes both private and combined settings.

Wells, *et al*,[22] suggest that mental health visits to general health providers commonly involve the treatment of both physical and mental health symptoms. An alternative explanation of this phenomenon is that contacts with the general health sector fulfill additional treatment needs, including social support and advice.

Patients' ambiguous definitions of psychiatric symptoms mixed with physical ailments may deter early diagnoses of mental health symptoms by the nonpsychiatric doctor or lead to misdiagnosis. Our study sample may reflect a tendency toward somatization. Economic strains combined with frequent unemployment as well as constant exposure to crime and violence make more flexible community standards of what constitute mental health problems. Such conditions could legitimize feelings of anxiety, depression, or sadness. The social acceptance of psychiatric symptoms may deter family, friends, and health professionals from referring needers to specialized mental health services.

This may be a consequence of the nonpsychiatric physician being gate-keepers of mental health care. Less than one-sixth (15 out of 103) of the specialty contacts were actually referred by physical health providers. Lack of referrals by nonpsychiatric physicians may be a major barrier to access to mental health services. Limited training in recognizing mental health symptoms by nonpsychiatric physicians or a negative attitude toward mental health care may impede referral.

Limitations of this study should be noted. The population surveyed cannot be assumed to be representative of all poor respondents in Puerto Rico or poor Puerto Ricans in the mainland. Poor persons not residing in low income areas of the island, as well as those in institutions (such as prison) or living in the streets, were excluded from this study. These findings cannot be extrapolated to poor Puerto Ricans in the mainland. Regional differences in Medicaid benefit coverage or local availability of services may influence the utilization rates for mental health services. Language barriers may also prevent access to services outside the island.

We cannot confirm self-reported utilization information provided by our respondents, therefore the validity of the information is not measured. It is probable that our measure of need (PSDS) identifies only certain areas of need for mental health services by the poor. We found that the PSDS scales do not detect as needers a high percentage of individuals who fulfill DIS criteria for alcohol abuse and dependence. However, we found a high concordance in the identification of needers by the CES-D, self-perception of mental health, and the PSDS.

Finally, we made empirical adjustments for the upward bias of poor physical health on the PSDS scale scores, although such empirical adjustment could underestimate actual need for mental health services among the poor. In our estimates of need, we sought to identify need for mental health services not due to physical health problems. The reliability of utilization data could be affected by elapsed time between actual use of services and the interview. Data from Kelly, *et al*,[33] suggest that reliability of medical information was not materially affected by the time interval between interviews. However, it was generally better when the time elapsed was less than one year, as was the case in our study.

In summary, the high utilization rate identified in our study group might be related to the organization of the public health care delivery system. The system in Puerto Rico is a vehicle of access to care for all in need, thus the coverage of the population is not constrained by the Medicaid income threshold standards as in the mainland United States. This broader coverage could be instrumental in explaining utilization patterns of Puerto Rico's low income population when compared to their counterparts on the mainland. Such comparisons can help assess the effects of organizational factors that influence or inhibit contact between low socioeconomic groups and mental health professionals. ☐

Acknowledgments

This research was supported by grant 5R01 MH4655 from the Division of Biometric and Clinical Applications, National Institute of Mental Health.

We would like to acknowledge Miguel Valencia, PhD, our sampling expert from the University of Puerto Rico; Hardeo Sahai, PhD, William Vega, PhD, and Philip Leaf, PhD, for their invaluable collaboration with this project.

References

1. Sheffler RM, Browne A: Demand analysis of mental health service use among ethnic subpopulation. Inquiry 1989; 26:202–215.
2. Meinhardt K, Vega W: A method for estimating underutilization of mental health services by ethnic groups. Hospital Community Psychiatry 1987; 38:1186–1190.
3. Karno M, Edgerton RB: Perception of

mental illness in a Mexican-American community. Arch Gen Psychiatry 1969; 20:233–238.

4. Padilla AM, Ruiz RA, Alvarez R: Community mental health services for the Spanish-speaking/surnamed population. Am Psychol 1975; 30:892–905.
5. Rogler LH: Help patterns, the family, and mental health: Puerto Ricans in the United States. Migration Rev 1978; 12:248–259.
6. Barrera M Jr: Mexican American mental health service utilization: A critical examination of some proposed variables. Community Ment Health J 1978; 14:35–45.
7. Treviño F, Moss A: Health Indicators for Hispanic, Black and White Americans. Vital and Health Statistics, Series 10, No. 148. Hyattsville, MD: NCHS, 1984.
8. Anderson R, Francis A, Licon J, *et al:* Psychologically related illness and health services utilization. Med Care 1977; 15:59–73.
9. McKinlay J: Some approaches and problems in the study of the use of services. J Health Soc Behav 1972; 13:115–152.
10. Kulka RA, Veroff J, Douvan E: Social class and the use of professional help for personal problems: 1957 and 1976. J Health Soc Behav 1979; 20:2–16.
11. Kadushin C: Why People go to Psychiatrists. New York: Atherton, 1969.
12. Gurin G, Veroff J, Feld S: Americans View Their Mental Health: A Nationwide Interview Survey. New York: Basic, 1960.
13. Hoppe SK, Heller PL: Alienation, familism and the utilization of health services by Mexican Americans. J Health Soc Behav 1975; 16:304–314.
14. Ware JE Jr, Young J: The measurement of health as a value. Carbondale, IL: School of Medicine, Southern Illinois University, 1976.
15. Johnston SA, Ware JE: Income group differences in relationships among survey measures of physical and mental health. Health Serv Res 1976; 11:416–429.
16. Regier DA, Goldberg ID, Burns BJ, Hankin JR, Hoeper EW, Nycz GR: Specialist/generalist division of responsibility for patients with mental disorders. Arch Gen Psychiatry 1982; 39:219–224.
17. Leaf PJ, Bruce ML, Tischler GL, Freeman DH, Weissman MM, Myers JK: Factors affecting the utilization of specialty and general medical mental health services. Med Care 1988; 26:9–26.
18. Wells KB, Manning WG, Duan N, Newhouse JP, Ware J: Sociodemographic factors and the use of outpatient mental health services. Med Care 1986; 24:75–85.
19. Regier DA, Goldberg ID, Taube CA: The de facto US mental health services system. Gen Psychiatry 1978; 35:685–693.
20. Shapiro S, Skinner EA, Kessler LG, Vonkorff M, German PS, Tischler GL, Leaf PJ, Benham L, Cottler L, Regier DA: Utilization of health and mental health services: Three epidemiological catchment area sites. Arch Gen Psychiatry 1984; 41:971–978.
21. Hough R, Landsverk J, Karno M, Burnam A, Timbers DM, Escobar JI, Regier DA: Utilization of health and mental health services by Los Angeles Mexican Americans and non-Hispanic whites. Arch Gen Psychiatry 1987; 44:702–709.
22. Brown G, Harris T: Life Events and Illness. New York: Guilford Press, 1989.
23. Belle D: Poverty and women's mental health. Am Psychol 1990; 45:385–389.
24. Taube CA, Rupp A: The effect of Medicaid on access to ambulatory mental health care for the poor and near-poor under 65. Med Care 1986; 24:677.
25. Bureau of the Census: Poverty thresholds in 1989, by size of family and number of related children. Washington, DC: US Department of Commerce, 1990.
26. Fuller WA, Kennedy W, Schnell D, Sullivan G, Park HJ: PC Carp version 1.0. Ames, Iowa. Iowa State University, Statistical Laboratory, 1989.
27. Vega WA, Warheit GJ, Meinhardt K: Mental health issues in the Hispanic community: The prevalence of psychological distress. *In:* Vega WA, Miranda MR (eds): Stress and Hispanic Mental Health: Relating Research to Services Delivery. Rockville, MD: National Institute of Mental Health, 1985.
28. Warheit GJ, Vega WA, Auth JB, *et al:* Psychiatric symptoms and dysfunctions among Anglos and Mexican Americans: An epidemiological study in Research in Community and Mental Health, Vol. 5. London: JAI Press, 1985.
29. Warheit GJ, Vega WA, Shimizer D, *et al:* Interpersonal coping networks and mental health among four race-ethnic groups. J Community Psychol 1982; 10:312–324.
30. Warheit GJ, Vega WA, Buhl-Auth J: Mental health needs assessment approaches: Case for applied epidemiology. Prev Human Serv 1983; 2:9–34.
31. Stefl ME, Prosperi DC: Barriers to mental health service utilization. Community Ment Health J 1986; 21:167–178.
32. Wells KB, Manning WP, Duan N, *et al:* Cost-sharing and the use of general medical physicians for outpatient mental health care. Health Serv Res 1987; 22:1–17.
33. Kelly JP, Rosenberg L, Kaufman DW, Shapiro S: Reliability of personal interview data in a hospital based case-control study. Am J Epidemiol 1990; 131:79–90.

A Prospective Study of Hospitalization with Gallstone Disease Among Women: Role of Dietary Factors, Fasting Period, and Dieting

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ABSTRACT

Background: Dietary risk factors for the development of gallstones have not been clearly established. We analyzed data from a population-based prospective study to determine dietary risk factors for hospitalization with gallstone disease.

Methods: We evaluated the role of dietary constituents, fasting, and dieting on subsequent hospitalization with gallstone disease among 4,730 women, ages 25 to 74 years, who participated in the first follow-up of the first National Health and Nutrition Examination Survey. Baseline dietary variables were established through a 24-hour dietary recall and a medical history. Proportional hazards models were used to calculate the effects of dietary variables while controlling for baseline risk factors.

Results: After an average of 10 years follow-up, gallstone disease was confirmed by hospital records among 216 women who denied gallstone disease at the baseline examination. The hazard rate of hospitalization with gallstone disease increased with increasing overnight fasting period and with dieting. Intake of fiber showed a small protective effect. The effect of energy intake was significant only among women younger than age 50 years at baseline. Results were not affected by adjustment for known risk factors for gallstone disease or other dietary factors.

Conclusion: A long overnight fasting period, dieting, and low fiber intake may increase the risk of hospitalization with gallstone disease. (*Am J Public Health*. 1991;81:880–884)

Introduction

Cholecystectomy is one of the most common surgical procedures in the United States, ranking fifth among all nonobstetric, therapeutic hospital procedures. In 1987, cholelithiasis, the leading reason for cholecystectomy, was the first listed diagnosis for 522,000 hospital discharges.[1]

Differences in diet are an appealing explanation for the wide variation in the occurrence of gallstones in the United States and internationally.[2,3] Numerous clinical and epidemiological studies have examined potential dietary risk factors for gallstones.[4–14] Such risk factors have included, among others, cholesterol,[4–6] linoleic acid,[5–7] oleic acid,[5–7] total carbohydrates,[6,8] fat,[6,8] fiber,[8–10] calcium,[11] and energy intake.[12,13] However, the results of these studies have not been consistent. Most of the epidemiological studies have used cross-sectional or case-control designs. One prospective study of energy intake found a positive association[12] with gallstones and another found no relation.[13] After examining more than 50 foods and nutrients the only association with diet in a recent prospective study of 88,373 middle age women was a negative one with vegetable fat and protein.[14] Clinical studies have suggested that certain dietary patterns, such as weight loss dieting and prolonged fasting may increase gallstone risk.[15–17]

The first National Health and Nutrition Examination Survey (NHANES I) collected dietary information. Follow-up interviews averaging 10 years later allowed prospective evaluation of dietary factors on subsequent disease. We tested specific hypotheses regarding the roles of dietary constituents, fasting, and dieting on the occurrence of hospitalization with gallstone disease in the longitudinal study of this population.

Methods

Subjects and Study Design

Baseline data, including dietary information, were collected in 1971–75 among a national probability sample of the United States population.[18,19] Follow-up of this cohort occurred in 1982–84 for those ages 25 to 74 years at the baseline examination.[20] The current study is limited to women because of the small number of cases of gallstone disease among men. Analysis was restricted to 4,730 Black and White women after excluding loss to follow-up (1,032 women), self-reported gallstones at baseline (591 women), unavailability of confirmatory hospital records for self-reported diagnosis of gallbladder disease during the follow-up (363 women), and lack of baseline information on gallstone disease (21 women), diet (65 women) or body mass index (two women).

Hospitalization with gallstone disease in the follow-up was coded according to the Ninth Revision of the International Classification of Diseases with Clinical Modification (ICD-9-CM). Cases had hos-

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pital discharge diagnoses of cholelithiasis (ICD-9-CM 574) (193 cases) or cholecystitis (ICD-9-CM 575.0–575.1) plus cholecystectomy (ICD-9-CM, procedure 51.22) (23 cases). The first date of hospitalization was regarded as the date of occurrence of clinical gallstone disease. Remaining for analysis were 216 women with hospital confirmed gallstone disease and 4,514 women without evidence of gallstones. A more detailed description of the study cohort and diagnostic criteria has been published.[21]

Variables and Measurements

All independent variables were measured at the baseline examination. Nutrient and energy intake were estimated from a 24-hour recall interview.[18] Participants were asked what they ate and the respective time, by hour and minute, for the 24-hour period beginning at midnight of the day before the interview. Based on these times two variables were created: number of meals and snacks per day, and the duration of overnight fasting period. Total number of intakes at least one hour apart equaled the number of meals and snacks. Overnight fasting period was estimated by the interval between the time of the last meal or snack and the first meal or snack ((24 hours − time of last meal) + time of first meal), using a 24-hour clock. Persons who ate only once during the 24-hour period were assigned a fasting period of 24 hours. Daily nutrient and energy intake were determined using standardized food composition tables applied to the foods reported in the dietary interview.[22] Fiber consumption was based on a provisional table of dietary fiber composition of foods.[23]

Self-reported alcohol consumption, recreational and nonrecreational physical activity, and dieting were ascertained from the baseline medical history interview. Dieting was based on the question "Are you on a special diet?"

Data Analysis

Dietary variables analyzed were those shown in Table 1 plus dieting. All of these variables have been postulated as having potential gallstone promoting activity.[4–13] Overnight fasting period was analyzed both as a continuous variable and as indicator variables (less than eight hour, eight to less than 10 hours, 10 to less than 12 hours, 12 to less than 14 hours, and at least 14 hours) to determine if there was a particularly increased risk with long fasting as has been suggested from clinical studies.[24]

TABLE 1—Medians, 25th and 75th Percentiles for Dietary Risk Factors for Women at the Baseline Examination, NHANES I, 1971–75

Factors	25th	Median	75th
Overnight fasting period (hr)	10.0	11.5	13.5
Number of meals (per day)	3.0	4.0	5.0
Energy (kcal)	1062	1412	1814
Carbohydrates (g)	115	160	210
Fat (g)	39	56	77
Saturated fat (g)	13	19	28
Oleic acid (g)	14	21	30
Linoleic acid (g)	3.0	5.4	8.9
Cholesterol (mg)	135	243	423
Calcium (mg)	287	503	790
Fiber (g)	4.8	7.5	11.2

In accordance with a previous analysis, non-dietary variables included were age in years and body mass index (weight in kg/height in m^2) as continuous variables, Black or White race, and parity categorized as at least four live births or less than four live births.[21] We also included as covariates alcohol consumption (g/day)[25] as a continuous variable and an index of physical activity. This last variable was constructed by summing the self-reported recreational and nonrecreational activities, each categorized in three levels.

Multivariate analysis used proportional hazards survival models with confirmation of the proportional hazards assumption of relatively constant rate ratios during the follow-up.[26] The effect of each dietary variable on the risk of gallstone disease was evaluated controlling for the non-dietary covariates: age at the baseline examination, body mass index (BMI), parity, race, quadratic terms for age and for BMI (age times age and BMI times BMI), and an interaction term for age and race (age times race). Interaction terms between the dietary variables and age, race, and BMI were examined for each dietary factor and were kept in the model if they improved significantly (p value < 0.05) the likelihood ratio of the model.[27] If an age interaction was present, the analysis was divided between persons less than age 50 and at least 50 years of age to simplify the models. Adjustment for energy consumption was made by including the 24-hour energy intake. For models including all ages, an interaction term of energy intake and age was added.

There were 65 women with no information on dieting and very few women (fewer than 13) with missing values for the other variables. Univariate analyses included all available information and multivariate analysis excluded persons with any missing values.

Results

Eighty-two percent of the women reported that what they ate during the 24-hour recall was the way they usually ate. Medians and the 25th and 75th percentiles are shown for each dietary variable at baseline (Table 1). Fifteen percent of women reported being on a special diet.

Survival analysis showed that overnight fasting, dieting, and fiber consumption were significantly related to gallstone disease (p value ≤ 0.03) when each dietary factor was adjusted for body mass index, body mass index^2, age, age^2, race, race times age, and parity. Number of meals, and total carbohydrate intake were marginally related to gallstone disease ($0.05 <$ p value < 0.10). Age influenced the effects of energy and fat intake (Table 2). Hazard rate ratios and 95 percent confidence intervals unadjusted and adjusted for energy intake are shown in Table 3 for those dietary factors with p values ≤ 0.10 in Table 2. Since the effects of energy intake and fat consumption were dependent on age, these variables were evaluated in separate models for women under and over age 50 years at the baseline examination. Energy adjustment reduced the hazard rate ratio for all variables.

When overnight fasting period was divided into five discrete periods the trend for an increased hazard ratio with increased overnight fasting was similar to the exponential model with overnight fasting as a continuous variable. Risk was greatest for 14 or more hours fasting, intermediate for 8–14 hours, and the least for fasting less than eight hours (Figure 1).

Additional analyses that included the significant dietary predictors of gallstone disease (dieting, overnight fasting period, fiber and energy intake), alcohol intake, and physical activity plus the other non-

TABLE 2—Hazard Coefficients* and Standard Errors (SE) for Dietary Factors for Hospitalization with Gallstone Disease for Women, NHANES I Epidemiologic Follow-up Study, 1971–84

Factors	Hazard Coefficient	SE	p value
Overnight fast period (hr)	0.078	0.024	0.003
Number of meals (per day)	−0.117	0.065	0.07
Dieting (yes/no)	0.513	0.158	0.001
Energy (kcal)	−0.0012	0.00041	0.02
Energy × age	0.00002	0.000007	0.007
Carbohydrates (g)	−0.0017	0.0010	0.08
Fat (g)	−0.0223	0.0080	0.006
Fat × age	0.0004	0.0001	0.01
Saturated fat (g)	−0.0057	0.0058	0.32
Oleic acid (g)	−0.0070	0.0057	0.22
Linoleic acid (g)	−0.0244	0.014	0.10
Cholesterol (mg)	−0.00016	0.00029	0.58
Calcium (mg)	−0.000006	0.0001	0.97
Fiber (g)	−0.029	0.014	0.03

*Each dietary variable evaluated individually adjusting for body mass index, body mass index^2, age, age^2, race, age times race, and parity.

TABLE 3—Hazard Ratios (HR)* and 95% Confidence Intervals (CI) for Dietary Factors for Hospitalization with Gallstone Disease among Women, NHANES I Epidemiologic Follow-up Study, 1971–84

		Unadjusted for energy intake		Adjusted for energy intake	
	Age	HR	95% CI	HR	95% CI
Energy (kcal)†	<50	0.69	0.53, 0.88		
	≥50	1.08	0.85, 1.36		
Overnight fasting period (hr)	all	1.33	1.10, 1.60	1.29	1.09,1.53
Dieting (Yes/No)	all	1.67	1.21, 2.23	1.66	1.21, 2.26
Number of meals/day	all	0.79	0.61, 1.02	0.83	0.64, 1.08
Carbohydrates (g)	all	0.85	0.70, 1.02	0.92	0.74, 1.34
Fat (g)†	<50	0.73	0.56, 0.95	1.08	0.62, 1.86
	≥50	1.07	0.88, 1.29	1.04	0.67, 1.60
Linoleic acid (g)	all	0.86	0.73, 1.02	1.10	0.90, 1.33
Fiber (g)	all	0.82	0.69, 0.97	0.85	0.70, 1.02

*All variables also adjusted for age, age squared, race, body mass index, body mass index squared, parity, and age × race. Models including all ages were also adjusted for energy × age. Except for diet, which was dichotomous, hazard ratios were obtained from models using continuous variables. Calculations were based on:

$$HR = e^{\text{hazard coefficient} \times (\text{75th–25th percentiles})}$$

†Variables with age interaction were analyzed by separate models for age groups: < 50 and ≥ 50 years.

dietary covariates specified in Table 3 did not substantially change any of the results (data not shown). Also, a model using as cases all women who reported hospitalization for gallbladder disease whether or not records were available gave essentially the same results (data not shown).

Discussion

In this prospective study of hospitalization with gallstone disease, a long overnight fasting period and dieting emerged as the two most important dietary risk factors for hospitalization with gallstone disease. Few studies have examined these risk factors. In two small, cross-sectional studies a positive association was found between duration of overnight fasting and gallstones.[11,28] A Danish population-based study of ultrasound diagnosed gallbladder disease found a history of "slimming treatments" associated with gallstones in men but not in women.[29] It was not known in the current study what proportion of women reporting a special diet were losing weight.

Long fasting period and dieting may act through diminished gallbladder motility, changes in the bile composition, or both to increase the risk of gallstones. Diverse conditions such as pregnancy,[30] parenteral nutrition,[31] diabetes,[32] and Chagas Disease[33] decrease gallbladder emptying and are considered risk factors for cholelithiasis. A clinical study of eight patients found that gallbladder contractions were reduced at mealtime during weight reduction, and the effect was more marked during prolonged fasting.[34] In addition, saturation of bile with cholesterol increases with fasting[24,35] and with weight loss.[36]

A recent study suggests that increased meal frequency may have a beneficial effect on health.[37] We did not find a strong relationship of number of meals and hospitalization with gallstone disease, but did find that a prolonged overnight fasting period increased the risk. Although fasting period was not a variable conceived when the original data were collected and edited, it is plausible that the times for meals and snacks reported on the 24-hour recall may represent usual eating times because of the regularity of daily activities for most persons. Longest fasting period based on 24-hour recall was also used in a Swedish study of diabetes,[38] but, as far as we know, it has not been validated, although 82 percent of the NHANES I study cohort considered the 24-hour recall their usual way of eating. Also, the increased risk after more than 14 hours of fasting resembles findings of bile composition studies in which lithogenicity of bile occurred only after a long period of fasting. Williams, *et al*, studied women without gallstones and found that saturation of bile with cholesterol was present in 4.5 percent after nine hours of fasting and in 54.5 percent after 16 hours of fasting.[24]

For the analyses based on the dietary composition, associations of gallstone disease hospitalization were found with low energy consumption among the younger group of women and women with low fiber intake. Other epidemiological studies have found negative,[8] positive,[12] and no relationship[13] between energy intake and gallbladder disease. The study that found a positive relationship was most similar to the current study, being a large, prospective study with extensive dietary data and an almost similar case definition. The negative association for energy intake that we found among younger women could be the result of a bias in the dietary recall or of differential energy expenditure between cases and noncases. Another possibility is

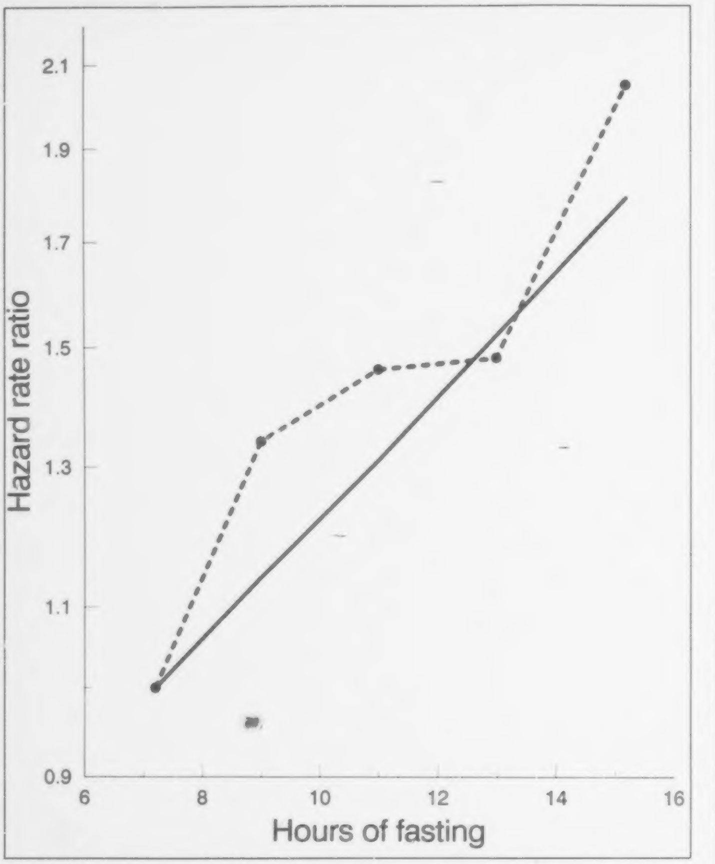

FIGURE 1—Duration of Overnight Fasting Period and Subsequent Hospitalization with Gallstone Disease in Women, NHANES I Epidemiologic Follow-up Study, 1971–84

The solid line represents the hazard rate ratio for overnight fasting period modeled as a continuous variable, compared with fewer than eight hours. Each point on the dashed line is the hazard rate ratio for ranges of overnight fasting period (8–10, 10–12, 12–14, and at least 14 hours) compared with fewer than 8 hours.

that women who have a propensity to form gallstones could have a higher metabolic efficiency and therefore lower energy intake that is independent of BMI.

There are also conflicting reports on the role of dietary fiber in gallstone disease.[9,10] Fiber is a convenient term for heterogeneous groups of poorly digested plant components whose nutritive functions are not well characterized. Colonic metabolism of fiber may affect bile secretion[39] and bind with bile acids in the gastrointestinal tract.[40]

Our results could not be explained by selective exclusion of cases without hospital records because analyses of all reported hospitalization for gallbladder disease, independent of availability of hospital records, showed similar results. Also, the prospective data collection eliminated the possible recall bias that is an unavoidable problem in case-control studies. Biased ascertainment could have occurred in this study if the risk factors analyzed increased the risk of gallstone symptoms rather than increase the risk of gallstones. However, a population-based study using ultrasonography showed that the frequency of dyspeptic symptoms was not different between women with and without gallstones.[41] Misclassification could have occurred in this study through the inclusion of asymptomatic cases of gallstones among the non-case group and the inaccuracies inherent in dietary interview.[42,43] In both cases the influence would be toward a lack of association between the dietary factors studied and gallstone disease.

Our most important findings were the independent roles of fasting and dieting in the development of gallstones. We also found associations with energy intake and dietary fiber. Methodological constraints did not permit absolute rejection of the hypothesis that other nutrients affect gallstone disease hospitalization. □

Acknowledgments

The authors wish to thank Danita Holt and Hermann Liau for data management and Elaine Lanza for creating the fiber data set.

References

1. Graves EJ: National Hospital Discharge Survey: Annual Summary, 1987. National Center for Health Statistics. Vital Health Stat 1989; 13(99).
2. Pixley F: Epidemiology. *In:* Bateson MC (ed): Gallstone Disease and Its Management. Boston: MTP Press Limited, 1986; 1–23.
3. Maurer KR, Everhart JE, Ezzati TM, *et al:* Prevalence of gallstone disease in Hispanic populations in the United States. Gastroenterology 1989; 96:487–492.
4. Lee DWT, Gilmore CJ, Bonorris G, *et al:* Effect of dietary cholesterol on biliary lipids in patients with gallstones and normal subjects. Am J Clin Nutr 1985; 42:414–420.
5. Scragg RKR: Aetiology of cholesterol gallstones. *In:* Bateson MC (ed). Gallstone Disease and Its Management. Boston: MTP Press Limited, 1986; 25–55.
6. Low-Beer TS: Nutrition and cholesterol gallstones. Proc Nutr Soc 1985; 44:127–134.
7. Sturdevant RAL, Pearce ML, Dayton S: Increased prevalence of cholelithiasis in men ingesting a serum-cholesterol-lowering diet. N Engl J Med 1973; 288:24–27.
8. Smith DA, Gee MI: A dietary survey to determine the relationship between diet and cholelithiasis. Am J Clin Nutr 1979; 32:1519–1526.
9. Scragg RKR, McMichael AJ, Baghurst PA: Diet, alcohol, and relative weight in gallstone disease: A case-control study. Br Med J 1984; 288:1113–1119.
10. Diehl AK, Haffner SM, Knapp JA, Hazuda HP, Stern MP: Dietary intake and the prevalence of gallbladder disease in Mexican Americans. Gastroenterology 1989; 97:1527–1533.
11. Williams CN, Johnston JL: Prevalence of gallstones and risk factors in Caucasian women in a rural Canadian community. Canad Med Assoc J 1980; 120:664–668.
12. Maclure KM, Hayes KC, Colditz GA, *et al:* Weight, diet, and the risk of symptomatic gallstones in middle-aged women. N Engl J Med 1989; 321:563–569.
13. Friedman GD, Kannel WB, Dawber TR: The epidemiology of gallbladder disease: Observations in the Framingham study. J Chronic Dis 1966; 19:273–292.
14. Maclure KM, Hayes KC, Colditz GA, Stampfer MJ, Willett WC: Dietary predic-

tors of symptom-associated gallstones in middle-aged women. Am J Clin Nutr 1990; 52:916–922.

15. Broomfield PH, Chopra R, Sheinbaum RC, *et al:* Effects of ursodeoxycholic acid and aspirin on the formation of lithogenic bile and gallstones during loss of weight. N Engl J Med 1988; 319:1567–1572.
16. Liddle RA, Goldstein RB, Saxton J: Gallstone formation during weight-reduction dieting. Arch Intern Med 1989; 149:1750–1753.
17. Pattinson NR, Chapman BA: Distribution of biliary cholesterol between mixed micelles and nonmicelles in relation to fasting and feeding in humans. Gastroenterology 1986; 91:697–702.
18. National Center for Health Statistics: Plan and Operation of the Health and Nutrition Examination Survey, United States, 1971–73. Hyattsville, MD: NCHS, 1977 (Vital and Health Statistics, series 1, no. 10a and 10b) (DHEW Pub. No. (PHS) 79-1310).
19. National Center for Health Statistics: Plan and Operation of the NHANES I Augmentation Survey of Adults 25–74 years, United States, 1974–75. Hyattsville, MD: NCHS, 1978 (Vital and Health Statistics, series 1, no. 14) (DHEW Pub. No. (PHS) 78-1314).
20. National Center for Health Statistics: Plan and Operation of the NHANES I Epidemiologic Follow-up Study 1982–84. Hyattsville, MD: NCHS, 1987 (Vital and Health Statistics, series 1, no. 22) (DHEW Pub. No. (PHS) 87-1324).
21. Sichieri R, Everhart, JE, Roth HP: Low incidence of hospitalization with gallbladder disease among Blacks in the United States. Am J Epidemiol 1990; 131:826–835.
22. Watt BK, Merrill AL: Composition of Foods: Raw, Processed, Prepared. Washington, DC: Department of Agriculture, 1963.
23. Lanza E, Butrum RR: A critical review of food fiber analysis and data. J Am Diet Assoc 1986; 86:732–743.
24. Williams CN, Morse JWI, MacDonald IA, Kotoor R, Riding MD: Increased lithogenicity of bile on fasting in normal subjects. Dig Dis Sci 1977; 22:189–194.
25. Schatzkin A, Jones DY, Hoover RN, *et al:* Alcohol consumption and breast cancer in the epidemiologic follow-up study of the first National Health and Nutrition Examination Survey. N Engl J Med 1987; 316:1169–1173.
26. Harrell FE: The PHGLM procedure. SUGI Supplemental Library User's Guide, version 5 ed. Cary, NC: SAS Institute, 1986.
27. Kleinbaum DG, Kupper LL, Morgenstern H: Epidemiologic Research. Belmont, CA: Lifetime Learning Publications, 1982.
28. Capron JP, Delamarre J, Herve MA, Dupras JL, Poulain P, Descombes P: Meal frequency and duration of overnight fast: A role in gallstone formation? Br Med J 1981; 283:1435.
29. Jorgensen T: Gallstones in a Danish population. Relation to weight, physical activity, smoking, coffee consumption and diabetes mellitus. Gut 1989; 30:528–534.
30. Everson GT, McKinely C, Lawson M, Johnson M, Kern F Jr: Gallbladder function in the human female: Effect of the ovulatory cycle, pregnancy and contraceptive steroids. Gastroenterology 1982; 82:711–719.
31. Roslyn JJ, Pitt HA, Mann LL, Ament ME, DenBesten L: Gallbladder disease in patients on long-term parenteral nutrition. Gastroenterology 1983; 84:148–154.
32. Stone BG, Gavaler JS, Belle SH, *et al:* Impairment of gallbladder emptying in diabetes mellitus. Gastroenterology 1988; 95:170–176.
33. Rocha A, Almeida HO, Teixeira VPA, Silva AM: Prevalência de colelitiase em chagásicos cronicos necropsiados no triângulo mineiro—correlação com o megaesofago, o megacólon e a insuficiência cardíaca. Arq Gastroenterol S Paulo 1985; 22:3–6.
34. Jacob BG, Richter WO, Schwandt P: Ursodeoxycholic acid and gallstones during weight loss [letter]. N Engl J Med 1989; 320:1352.
35. Metzger AL, Adler R, Heymsfield S, Grundy SM: Diurnal variation in biliary lipid composition: Possible role in cholesterol gallstone formation. N Engl J Med 1973; 288:333–336.
36. Bennion LJ, Grundy SM: Effects of obesity and caloric intake on biliary lipid metabolism in man. J Clin Invest 1975; 56:996–1011.
37. Jenkins DJA, Wolever TMS, Vuksan V, *et al:* Nibbling versus gorging: Metabolic advantages of increased meal frequency. N Engl J Med 1989; 321:929–934.
38. Lundgren H, Bengtsson C, Blohme G, *et al:* Dietary habits and incidence of noninsulin-dependent diabetes mellitus in a population study of women in Gothenburg, Sweden. Am J Clin Nutr 1989; 49:708–712.
39. Heaton KW: Gallstones. *In:* Spiller GA (ed): Handbook of Dietary Fiber in Human Nutrition. Boca Raton, FL: CRC Press, 1986; 361–367.
40. Lee SP: Diseases of the liver and biliary tract. *In:* Kinney JM, Jeejeebhoy KN, Hill GL, Owen OE (eds): Nutrition and Metabolism in Patient Care. Philadephia: WB Saunders Company, 1988; 313–341.
41. GREPCO—The Rome Group for Epidemiology and Prevention of Cholelithiasis: Prevalence of gallstone disease in an Italian adult population. Am J Epidemiol 1984; 119:796–805.
42. Block G: A review of validation of dietary assessment methods. Am J Epidemiol 1982; 115:492–505.
43. Beaton GH, Milner BA, Corey P, *et al:* Sources of variance in 24-hour dietary recall data: Implications for nutrition study design and interpretation. Am J Clin Nutr 1979; 32:2546–2559.

Cervical Cancer Screening: Who Is Not Screened and Why?

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ABSTRACT

Background: The decline in death rates from cervical cancer in the United States has been widely attributed to the use of Papanicolaou (Pap) smears for early detection of cervical cancer.

Methods: Pap smear screening rates, beliefs about appropriate screening intervals and factors affecting screening were examined using 1987 National Health Interview Survey data.

Results: Results indicate that through age 69, Blacks are screened at similar or higher rates than Whites. Hispanics, particularly those speaking only or mostly Spanish, are least likely to have received a Pap smear within the last three years. Of women who had never heard of or never had a Pap smear, nearly 80 percent reported contact with a medical practitioner in the past two years, while more than 90 percent reported a contact in the past five years. Overall, the most frequently reported reason for not having a recent Pap smear was procrastinating or not believing it was necessary.

Conclusions: Thus, in developing screening programs, Hispanics, particularly Spanish speakers, must be targeted. In addition, educational programs should target unscreened women who forego the test due to underestimating its importance, procrastination, or because their medical care provider did not suggest the procedure. Women must be intensively educated that Pap smears should be scheduled routinely to detect asymptomatic cervical cancer. (*Am J Public Health.* 1991;81:885–891)

Introduction

Although cervical cancer is a readily curable disease if diagnosed and treated early, survival rates have not continued to improve over the mid-1970s and early 1980s.[1] Although these static survival rates may be related to better survivors being diagnosed in the *in situ* stage while women with poorer survival potential are diagnosed later, improved screening and early detection could increase survival rates and lower mortality. In addition, while overall rates of Pap smear screening are reasonably high, a significant number of women are not being screened.

Using the 1973 and 1985 National Health Interview Survey (NHIS), Makuc, *et al*, described changes in use of Pap smears by United States women.[2] They reported that poor women were less likely to use preventive services than nonpoor. They also found that between 1973 and 1985 Black women had substantially increased their use of Pap smears with rates exceeding those of White women. Older women also increased their screening rates, but their rates continued to be lower than younger women.

Using data from a telephone survey, Hayward, *et al*, found, in a multiple regression analysis, age was inversely related to the use of Pap smears.[3] Having health insurance, a higher level of education, being employed, and Black race (compared to Whites and Hispanics combined) were positively related to the use of Pap smears.

In a study conducted by 15 state health departments and the District of Columbia, Parker reported that 72 percent of the female participants had a Pap smear within the last year.[4] Rates decreased with increasing age, were higher in Blacks than in non-Hispanic Whites, and rose with increasing income. In this study, only 1 percent of the women had never had a Pap smear and 0.2 percent had never heard of the test. The knowledge and previous screening rates are much higher than other published rates.[2,3]

In a case-control study of cervical cancer patients, Celentano found that cases were significantly less likely than controls to have ever had a Pap smear or to have had regular Pap smears.[5] Cases were also older at the first Pap smear and more frequently reported not being informed that they should have routine Pap smears.

In England, a general medical practice studied women overdue for Pap smears.[6] Of the 118 women personally contacted, 47 percent said they had no major objection to the test, but it was of low priority. Another 24 percent gave reasons for not having the procedure which were based on incorrect information and 29 percent had strong objections to the test and would probably never consent to having a Pap smear.

Although research suggests Pap smear screening is relatively common, there are subgroups which remain resistant to screening efforts. The literature does not detail the underlying reasons for this failure. The 1987 National Health Interview Survey (NHIS) data offers an opportunity to explore these reasons. This paper will identify subgroups failing to utilize appropriate screening, as well as their underlying rationale.

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TABLE 1—Unweighted Number and Weighted Percent ( ) of Women Responding to 1987 NHIS Cancer Control Supplement by Race and Age

Age (years)	White	Black	Hispanic	Other	
18–29	2223 (24.3)	544 (31.3)	295 (37.4)	98 (37.9)	
30–39	2086 (21.2)	467 (22.0)	214 (22.0)	83 (26.5)	
40–49	1374 (15.6)	279 (16.6)	124 (15.3)	29 (11.0)	
50–59	1126 (12.9)	230 (12.5)	83 (11.6)	26 (13.3)	
60–69	1346 (12.8)	199 (9.4)	81 (8.5)	17 (7.1)	
70+	1678 (13.1)	199 (8.1)	51 (5.2)	16 (4.2)	
Total	9833 (99.9)	1918 (99.9)	848 (100)	269 (100)	12868

TABLE 2—Proportion of Women Never Having Heard of a Pap Smear and Proportion Compliant with Screening by Age and Race

	White				Black				Hispanic			
	Never Heard		Compliant		Never Heard		Compliant		Never Heard		Compliant	
Age (years)	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.
18–29	2.6	(0.5)	83.3	(1.1)	3.4	(1.0)	85.3	(2.4)	16.0	(2.1)	67.2	(4.0)
30–39	0.6	(0.2)	85.7	(0.8)	1.4	(0.5)	90.8	(1.5)	10.0	(1.8)	76.9	(3.0)
40–49	2.0	(0.5)	74.9	(1.3)	0.5	(0.3)	89.1	(2.3)	10.3	(2.6)	75.5	(3.8)
50–59	0.6	(0.2)	72.0	(1.5)	4.5	(1.5)	70.3	(3.4)	19.7	(5.1)	53.6	(6.5)
60–69	1.7	(0.4)	61.6	(1.6)	5.1	(2.0)	61.1	(4.4)	19.4	(5.2)	43.4	(8.6)
70+	5.9	(0.7)	44.9	(1.4)	19.7	(3.6)	34.0	(3.8)	26.8	(6.4)	29.7	(6.2)
Total	2.1	(0.2)	73.3	(0.5)	4.1	(0.6)	78.9	(1.2)	15.1	(1.1)	65.0	(2.3)

Methods

Data from the 1987 NHIS were used to examine factors associated with the use of Pap smears by women in the United States. NHIS is a nationwide survey conducted by the National Center for Health Statistics (NCHS). When appropriate statistical methods are applied, estimates are representative of the US noninstitutionalized, civilian population. The 1987 sample design provided for an oversampling of Blacks and the Hispanic population directed at obtaining better estimates for these populations.

In addition to the core questions administered by NCHS, two supplemental questionnaires were added for the National Cancer Institute (NCI). One questionnaire focused on epidemiologic aspects of cancer, such as dietary intake. The second questionnaire addressed issues on cancer control, attitudes, and use of cancer screening. One of the two questionnaires was administered to one randomly selected adult member (aged 18 or over) of each of the approximately 44,000 households; 22,043 individuals completed the Cancer Control Supplement.

The NHIS Cancer Control Supplement included questions about the use of several cancer screening tests. With respect to Pap smears, women were asked, "Have you ever heard of a Pap smear test?" If a woman had not, she was not questioned further. Those women having knowledge of Pap smears were asked if they had ever had a Pap smear and when they last had one. Additional questions were asked about use for health problems, test results, the primary reason for not having the test, method used to advise them of results, and menstrual status, including hysterectomies.

Compliance was defined as having had a Pap smear in the past three years. The NCI's Working Guidelines suggest that after three or more consecutive normal annual Pap smears, the test may be performed less frequently at the discretion of the physician. Three years, often recommended as the appropriate interval, was chosen for the purpose of these analyses.[7]

Reasons for non-compliance were categorized into seven groups: 1) put off (the woman thought the Pap smear was unnecessary; she was simply putting it off or she had no problems and therefore did not have the test); 2) hysterectomy (the woman states she had a hysterectomy and therefore did not have a Pap smear); 3) cost (includes cost and having no insurance); 4) no MD (had no physician); 5) not recommended (the health care provider did not suggest a Pap smear or had said it was unnecessary); 6) embarrassment or fear on the part of the woman, or 7) other or unknown.

Totals, age-specific means and age-adjusted means were calculated using the SESUDAAN procedure.[8] This procedure takes into account the complex multistage sampling of the NHIS study. The RTI-LOGIT program was used for multi-variate logistic regression analysis taking into account the sample design.[9]

Results

The NHIS Cancer Control questionnaire was completed by 12,868 women age 18 and older. The unweighted distribution and weighted (to 1987 U.S. Census estimates) percentage of women by age and race can be found in Table 1. Women, other than non-Hispanic Blacks, non-Hispanic Whites or Hispanics, were excluded from further analyses because of the small sample size (n = 269). In addition, 3.6 percent of the women were unable to recall the date of their last Pap smear. These women were excluded from the analyses.

Age, Race, and Ethnicity

When the proportions of women never having heard of Pap smear are compared by age and race, the results are rather dramatic (Table 2). Many more Hispanic women have never heard of a Pap smear than either Black or White women. The proportion of women under the age of 70 having never heard of a Pap smear was 2.6 percent for White, 5.1 percent for Black, and 19.7 percent for Hispanic women.

As expected, compliance with screening is lower in Hispanics than for either Blacks or Whites (Table 2). Through age 29, compliance rates are similar for Blacks and Whites. From age 30 through 49, however, Blacks are significantly more compliant. In women age 70 and above, compliance rates for White

TABLE 3—Age-Adjusted Percent of Women Never Having Heard of or Interval Since Last Pap Smear by Primary Language Spoken

Language Spoken	Never Heard	Never Had	Last Pap Smear <3 yrs	Last Pap Smear >3 yrs
Mostly Spanish	34.3 (5.0)	8.7 (2.6)	48.0 (6.5)	8.9 (2.8)
Both Equally	7.3 (3.6)	11.2 (3.7)	60.7 (5.0)	20.8 (4.2)
Mostly English	6.9 (2.7)	8.2 (2.5)	72.4 (3.5)	12.5 (2.9)

TABLE 4—Age-Adjusted Percent of Women Never Having Heard of or Interval Since Last Pap Smears by Poverty Index, Education, and Usual Source of Care

Variable	Never Heard	Never Had	Last Pap Smear <3 yrs	Last Pap Smear >3 yrs
Poverty Index Ratio				
Below	6.2 (0.8)	10.9 (0.9)	64.8 (1.4)	18.1 (1.0)
At or above	2.5 (0.2)	6.5 (0.4)	75.2 (0.5)	15.9 (0.4)
Education				
<12 years	7.5 (0.6)	9.2 (0.8)	63.9 (1.2)	19.5 (0.8)
12 years	2.4 (0.3)	6.3 (0.5)	74.5 (0.8)	16.8 (0.6)
>12 years	1.5 (0.2)	6.2 (0.5)	79.0 (0.8)	13.3 (0.7)
Usual Source of Care				
HMO	1.1 (0.5)	4.7 (1.1)	85.2 (1.6)	8.9 (1.1)
Doctor's office	2.5 (0.2)	6.9 (0.4)	75.1 (0.6)	15.5 (0.4)
All other sources	4.7 (0.7)	6.5 (0.9)	73.7 (1.3)	15.1 (1.0)
No usual source	7.3 (0.9)	11.6 (1.0)	58.2 (1.4)	23.0 (1.3)
Total	3.3 (0.2)	7.2 (0.3)	73.3 (0.5)	16.1 (0.4)

women increase significantly over those for Black women. Through age 39, Hispanic women are less compliant than either Blacks or Whites. While above this age the rates vary greatly, the much lower rates for Hispanics suggest important differences in screening patterns.

To identify possible explanations for the low Hispanic compliance rate, this group was evaluated according to primary language: 1) only or mostly English (30.1 percent); 2) only or mostly Spanish (34.5 percent); or 3) both languages equally (32.2 percent) (undetermined 3.1 percent, excluded) (Table 3). When age-adjusted means were compared, nearly five times more Spanish speaking women had never heard of Pap smears as compared to English speakers. Much lower compliance rates for Spanish speakers were also apparent.

When compared by birthplace (US, Mexico, Cuba, and Puerto Rico or elsewhere), language appears to be a better predictor of screening compliance (data not shown). Using the self-categorization of Hispanic ethnic origin, Cubans were much less likely to be compliant than Puerto Ricans, Mexicans or "Other" Hispanics, who had similar rates of compliance. However, after adjusting for age, only "Other" Hispanics had significantly lower rates of compliance when compared to Mexicans. This may be important since "Other" Hispanics probably identifies women from South and Central America.

Other Variables

Rates of never having heard of Pap smear are over twice as high for women below the poverty index ratio, a measure of family income adjusted for household size (Table 4). When women above and below the poverty index are compared, it is apparent that the latter are less likely to have had a Pap smear in the past three years.

Education, as well as income, influences health behavior. Women with less than a high school education were three times as likely to have never heard of a Pap smear (Table 4). Women failing to complete high school were much more likely to be non-compliant with screening than those who graduated.

Menopausal status might also affect compliance rates. Women with non-surgical (primarily "natural") menopause were the least likely to be compliant. Premenopausal women were most likely to be screened in the last three years and women with surgical menopause (e.g. hysterectomies) fell between the two groups (data not shown). However, when rates are compared for these three groups after adjusting for age, no important differences in compliance are seen. Women who had undergone hysterectomies were included because excluding them did not appreciably alter other analytic results.

Attitudes toward cancer might influence use of screening Pap smears. When asked whether they could do very little to reduce their chances of getting cancer, women who agreed or strongly agreed were less likely to have had a Pap smear in the last three years than those who disagreed. Those who strongly disagreed were no more likely to have been screened in the last three years than women who disagreed. Women with "no opinion" were much less likely than any other category to have had a recent Pap smear.

For a variety of reasons—such as out-of-pocket expense, attitude of provider or setting where care is given—source of care might influence compliance with screening. Women in health maintenance organizations (HMOs) or other prepaid medical plans were more likely to have been screened within the last three years.* More than one-fourth of the women who reported doctor's office care were non-compliant. This is important since 70 percent of all women reported a physician's office as their primary source of care.

Timing of Pap Smears

Beliefs regarding proper screening intervals would also be expected to alter the frequency. When asked to describe appropriate screening intervals, responses ranged from "never" or "when problems occur" to "more frequently than once per year." Women who were unfamiliar with the procedure were not queried. Of those familiar, 77.9 percent described appropriate intervals as 13 to 36 months (one to three years) while 10.2 percent said annually (data not shown).

Whites were more likely than others to suggest a one- to three-year screening interval. However, Blacks and Hispanics were more likely to suggest a more frequent than annual screening. No important differences were identified among Hispanics when reported timing was examined by language spoken.

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*Bernstein AB, Thompson GB, Harlan LC: Differences in rates of cancer screening by usual source of medical care: Data from the 1987 National Health Interview Survey. (In press).

TABLE 5—Age-Adjusted Reported Reason for No Current Pap Smear by Race, Poverty Index, Education, Usual Source of Care, and Menopausal Status

	Put off		Hysterectomy		Cost		No MD		Not recommended		Embarrased Fear		Other/ Unknown	
	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.	$\bar{X}$	S.E.
Age (years)														
18–29	50.8	(3.6)	0.3	(0.3)	5.2	(1.3)	5.0	(1.4)	11.3	(1.9)	7.0	(1.7)	20.4	(2.7)
30–39	47.0	(2.9)	7.8	(1.7)	9.9	(1.7)	8.8	(1.7)	1.0	(0.6)	6.6	(1.7)	18.9	(2.1)
40–49	45.4	(3.3)	15.6	(2.1)	5.6	(1.2)	7.7	(1.8)	5.9	(1.7)	1.9	(0.9)	17.9	(2.6)
50–59	42.2	(2.9)	20.5	(2.6)	6.0	(1.6)	5.7	(1.3)	8.8	(1.7)	1.2	(0.8)	15.5	(1.9)
60–69	50.2	(2.5)	14.1	(1.6)	3.2	(0.8)	6.6	(1.2)	10.8	(1.5)	2.3	(0.6)	12.9	(1.7)
70+	46.6	(2.0)	10.4	(1.2)	0.6	(0.3)	2.1	(0.5)	22.1	(1.8)	1.6	(0.4)	16.5	(1.8)
Race														
White	46.9	(1.5)	10.8	(0.8)	5.8	(0.7)	6.3	(0.7)	9.0	(0.9)	3.8	(0.7)	17.4	(1.0)
Black	49.9	(3.6)	2.8	(0.9)	6.1	(1.7)	6.1	(2.2)	13.1	(2.6)	2.5	(1.1)	19.4	(2.7)
Hispanic	47.8	(4.3)	2.5	(1.6)	4.0	(1.2)	4.2	(2.4)	11.5	(2.6)	8.7	(3.0)	21.4	(3.8)
Poverty Index Ratio														
Below	41.0	(2.9)	6.9	(1.9)	11.3	(2.3)	6.6	(1.7)	11.7	(2.0)	3.7	(1.4)	18.9	(2.4)
At/Above	48.8	(1.5)	10.2	(0.7)	4.3	(0.6)	6.5	(0.9)	8.5	(0.9)	4.5	(0.8)	17.3	(1.1)
Education (years)														
<12	45.6	(2.3)	8.6	(1.0)	7.7	(1.5)	4.7	(1.1)	8.5	(1.3)	6.6	(1.9)	18.3	(2.3)
12	49.0	(2.0)	10.5	(1.1)	6.1	(1.2)	5.5	(0.7)	9.6	(1.3)	3.9	(0.9)	15.4	(1.4)
>12	46.7	(2.6)	10.2	(1.2)	3.4	(0.7)	7.8	(1.6)	8.2	(1.1)	2.7	(0.8)	20.9	(1.9)
Usual source of care														
Office	47.6	(1.6)	11.4	(0.9)	5.2	(0.7)	4.8	(0.8)	9.7	(1.0)	3.7	(0.8)	17.6	(1.2)
HMO	42.5	(7.0)	8.8	(3.5)	3.7	(2.1)	3.5	(2.5)	12.3	(5.9)	1.3	(1.3)	27.9	(7.2)
All other	46.8	(3.0)	6.4	(1.3)	6.3	(1.8)	6.6	(1.6)	7.9	(1.6)	4.6	(1.5)	21.4	(2.4)
No USC	49.2	(3.2)	5.1	(1.2)	7.0	(1.5)	11.9	(1.9)	6.5	(1.7)	5.3	(1.6)	15.0	(2.0)
Menopausal Status														
Pre	51.3	(4.2)	—		7.5	(1.8)	5.8	(1.0)	5.7	(1.6)	3.8	(0.7)	20.0	(3.6)
Surgical	37.7	(2.7)	4.3	(1.5)	2.9	(0.9)	2.3	(1.3)	12.8	(1.6)	1.5	(0.9)	38.4	(1.5)
Post	52.2	(7.6)	—		2.3	(0.9)	10.0	(5.6)	11.9	(5.9)	9.1	(6.2)	14.4	(4.1)
Total	47.5	(1.4)	9.7	(0.7)	5.6	(0.6)	6.2	(0.7)	9.2	(0.8)	4.2	(0.6)	17.8	(0.9)

Reasons Reported for No Screening

Among those familiar with but non-compliant, the primary reason for not having a Pap smear was examined. The largest category of reasons given by women for being non-compliant was they believed it was unnecessary, had no problems, or had been procrastinating (Table 5). This category accounted for nearly half of the reasons given.

Reasons for not having a Pap smear varied greatly by age. Compared to other age groups, women in the youngest and oldest two groups more frequently stated that the physician had not recommended the test. The two youngest age groups were more likely than others to suggest embarrassment or fear as the reason for non-compliance.

By race no important differences were noted after adjusting for age, except that Whites were more likely than Blacks and Hispanics to suggest that the reason for no current Pap smear was having had a hysterectomy. When the age-adjusted reasons for lack of compliance were compared, surgical postmenopausal women gave "not recommended by the physician" more frequently and "procrastination" less frequently than premenopausal women (Table 5). Cost was a much more important factor for premenopausal women.

Logistic Regression Analyses

To determine factors associated with having heard of a Pap smear or compliance with screening, separate analyses using a backwards, stepwise logistic regression were performed for each as dependent variables (Table 6). Independent variables included: age (continuous), age squared (continuous), marital status (married vs divorced/separated, widow, never married), primary source of care (doctors' office vs HMO, all other sources, and no usual source), place of residence (urban vs rural), education (<12 years vs 12 years, >12 years), self-reported health status, menopausal status, attitude/knowledge about risk reduction (disagree vs strongly agree, agree, strongly disagree, no opinion) and language spoken (English vs Spanish, both languages equally). These variables were selected based on review of literature and/or previous univariate analyses.

In the logistic regression model, age and age squared were important predictors of knowledge of Pap smears, indicating a non-linear relationship of age and knowledge. Women who spoke only or mostly Spanish were less likely to have ever heard of Pap smears than women who spoke only or mostly English. No differences were seen between women whose usual source of care was a physician's office and those with any other usual source of care. However, women with no usual source of care were less likely to have heard of Pap smears than women using doctors' offices for care. Women with less than 12 years of education and those from urban areas were less likely to have ever heard of Pap smears, as were those who strongly agreed, agreed, or had no opinion about the risk statement.

A multiple regression analysis with compliance with screening (no, yes) as the dependent variable was performed (Table 7). These models included all independent variables listed above. Age, although an important predictor of compliance, has an inverted U-shape association with compliance. Compliance appears to peak around age 50, decreasing beyond that age 50 to a point lower than in the youngest age group. Speaking Spanish as the primary language was associated with a lower compliance rate. Never married women were less likely than married women to be compliant. Women who stated their primary source of care as an HMO were more likely to be compliant and women with no source of care were less likely to be compliant than women using a doctor's office. Women with 12 or more years of education were more likely to be compliant.

TABLE 6—Beta Coefficients and Standard Errors for Predictors of Never Heard of Pap Smears

Independent Variables	Beta	STD Error
Langage		
Mostly Spanish speaking	2.2399*	0.2838
Both languages	−0.1058	0.5340
Age	−0.1260*	0.0195
Age^2	0.0012*	0.0002
Usual Source of Care		
HMO	0.1166	0.4498
All other	0.4743*	0.2286
No care	1.1200*	0.1669
Education		
12 years	−0.7891*	0.1438
>12 years	−0.9945*	0.1659
Attitude		
Strongly agree	0.5432	0.3311
Agree	0.7394*	0.1679
Strongly disagree	0.3771	0.2589
No opinion	1.7424*	0.1385
Urban	0.4319*	0.1782

*Significant at $p < 0.05$.

TABLE 7—Beta Coefficients and Standard Errors for Predictors of Having Had a Pap Smear within the Past 3 Years

Independent Variables	Beta	STD Error
Langage		
Mostly Spanish speaking	−0.9981*	0.3459
Both languages	−0.4605	0.2447
Age	0.0034	0.0080
Age^2	−0.0004*	0.0001
Marital Status		
Divorced/Separated	−0.0567	0.0684
Widow	−0.1123	0.0714
Never Married	−0.9366*	0.0721
Usual Source of Care		
HMO	0.3724*	0.1157
All other	0.0457*	0.0953
No care	−0.7428*	0.0748
Education		
12 years	0.3582*	0.0612
>12 years	0.6381*	0.0739
Attitude		
Strongly agree	−0.0353	0.1737
Agree	−0.1859*	0.0694
Strongly disagree	−0.1657*	0.0757
No opinion	−0.5627*	0.0672

*Significant at $p < 0.05$.

Discussion

Data here confirm the findings of Makuc, *et al*, that screening in Black women now exceeds screening in Whites.[2] These data also confirm that screening decreases with increasing age and increases with education.[2,3] Although the screening rates are higher for Blacks, the mortality rate is lower for Whites.[1] This may be due to the fact that only recently have screening rates increased above those for Whites. It is also possible that follow-up of positive Pap smears is not as complete for Black women.

Data suggest that Hispanic women are at an increased risk of mortality and morbidity from cervical cancer.* Despite this fact, it is clear that these women are not being adequately screened. Several factors appear to contribute to this lack of screening.

One might suspect that older Hispanic women would be less inclined to learn English and age would therefore confound the relationship between language spoken and compliance. Even after adjusting for age and other variables, women speaking only or mostly Spanish remained less likely to have been screened. Language spoken may be a surrogate measure for acculturation. There may be a cultural avoidance of medical tests such as Pap smears. Although the estimates were somewhat unstable because of small numbers, many more Spanish speakers (17.7 percent) than English speakers (1.5 percent) reported the primary reason for no current Pap smear was because of embarrassment or fear. However, over 11 percent (age-adjusted) of the Spanish speaking women reported they were non-compliant with screening because their physician had not suggested it or had said it was unnecessary. Also contributing may be the fact that few medical care providers are able to fully communicate in Spanish and therefore are unable to convey the importance and need for a Pap smear.

Non-compliant Hispanic women account for approximately 1.6 million unscreened women and Hispanics are the fastest growing segment of our population.[10] Any program to increase screening participation will ultimately need to address existing cultural differences, especially for Spanish speakers.

Usual source of medical care appears also to influence compliance. Women whose usual source of care was an HMO were more likely to be screened than women whose usual source of care was a physician's office. This is an important source of unscreened women since most women still have their primary source of care in a physician's office. Nationwide this would account for about 18.6 million women who were unscreened in the last three years. Some physicians who are the primary source of care for a woman may not provide Pap smear tests, e.g. the internist may think gynecologists should do the test, and a woman may not have regular gynecological care (data not available in this data set). However, screening would probably increase if these physicians provided referrals.

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*Horm JW: Cancer Incidence, Survival, and Mortality Among Racial and Ethnic Groups in the U.S. (unpublished).

Screening compliance might be affected by the manner in which the results are communicated. Makuc, *et al*, found that the rates for Pap smears in Blacks had increased substantially between 1974 and 1985, suggesting a targeted effort was made in this group.[3] As part of that effort of reinforcing compliance, physicians might be giving test results in person. Indeed, more than half of the Black women received their Pap smear results in this way. Women with less education and lower incomes were more likely to receive the results of their Pap smears in person; however, these women were less likely to have had a Pap smear within the last three years. In addition, women whose usual source of care was an HMO have the highest rate of Pap smear screening, but most commonly these women are not contacted with normal results. Even with diagnostic Pap smears, HMO-enrolled women were still more likely not to be contacted with normal results. These findings suggest that in-person results may instead reflect the provider concern that follow-up might be difficult when results are abnormal, but does not support the hypothesis that in-person results reinforce screening.

Knowledge of proper screening intervals would be expected to influence compliance with screening. When queried about the proper frequency of screening, 75 percent of the women believed the appropriate screening interval was one to three years. However, when examined by race, more Black and Hispanic women believed screening should be performed with a more than annual frequency. Despite the fact that Black women more often reported an inappropriate frequency for Pap smears, they were still more likely to be screened than White women. In addition, while Hispanic women were more likely to say that Pap smears should be performed more than annually, they were the least likely to have received them at the appropriate rate. This suggests components other than knowledge may be encouraging the screening of Black women, since knowledge does not necessarily translate into proper screening practice.

Reasons varied for non-compliance, but nearly 60 percent of the reasons basically revealed a lack of appreciation of the importance of screening. An estimated 12 million US women could be included in this group along with 2.5 million who have never heard of Pap smears. These findings suggest that this population is not adequately educated about the importance of routine screening, lack encouragement from medical providers, or have substantial misunderstanding about the messages from them regarding Pap smears.

Women may be getting mixed messages about screening, with the recommendations for screening frequency seemingly unclear. In addition, the controversy regarding the accuracy of the reading of Pap smears, which has been well reported in the lay press, might convey a message that screening is unimportant or results are of questionable quality.[11] A clear, unambiguous statement about the benefits and timing of Pap smears needs to be emphasized.

Lack of screening compliance can be attributed to multiple causes. Any effective screening intervention must include a multi-dimensional strategy that will successfully address these variables and influence behavior to generate better compliance among women at risk of developing cervical cancer. □

References

1. Ries LAG, Hankey BF, Edwards BK: Cancer Statistics Review 1973–87. Bethesda, MD: National Cancer Institute, 1990.
2. Makuc DM, Freid VM, Kleinman JC: National trends in the use of preventive health care by women. Am J Public Health 1989; 79:21–26.
3. Hayward RA, Shapiro MF, Freeman HE, Corey CR: Who gets screened for cervical and breast cancer? Results from a New National Survey. Arch Intern Med 1988; 148:1177–1181.
4. BRFSS Coordinators: Pap smear screening—Behavioral Risk Factor Surveillance System, 1988. MMWR 1989; 38:777–779.
5. Celentano DD, Klassen AC, Weisman CS, Rosenshein NB: Cervical cancer screening practices among older women: Results from the Maryland cervical cancer case-control study. J Clin Epidemiol 1988; 41:531–541.
6. Meadows P: Study of the women overdue for a smear test in a general practice cervical screening programme. J R Coll Gen Pract 1987; 37:500–503.
7. National Cancer Institute, Early Detection Branch, Division of Cancer Prevention and Control: Working Guidelines for Early Cancer Detection Rationale and Supporting Evidence to Decrease Mortality. 1987 (unpublished).
8. Shah BV: SESUDAAN: Standard errors program for computing of standardized rates from sample survey data. 1981 (unpublished).
9. Shah BV, Folsom RE, Harrell FE, Dillard CN: Survey data analysis software for logistic regression. 1984 (unpublished).
10. Horm JW, Asire AJ, Young JL, Pollack ES: Cancer Incidence and Mortality in the United States (SEER) 1973–81: Bethesda, MD: National Institutes of Health, 1984.
11. Hill R: Lab Accused of Misreading Pap Smears Closes in MD. Washington Post April 14, 1990; p. A2.

Public Health Briefs

Sustained Effects of an Educational Program to Reduce Sales of Cigarettes to Minors

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ABSTRACT

We report 1-year follow-up data from a sample of stores participating in a 6-month community-wide educational effort to reduce cigarette sales to minors in Santa Clara County, California. The proportion of over-the-counter sales to minors at the 1-year follow-up illustrated that although statistically significant reductions were maintained 6 months after the intervention ended, recidivism occurred. Suggestions for achieving long-term reductions in sales to minors are offered. (*Am J Public Health*. 1991;81:891–893)

Introduction

Although there are many factors that influence the decisions that adolescents make to initiate or continue smoking, one that has received only recent attention is access to tobacco. Few smokers begin smoking after their teenage years, and the earlier a young person begins using tobacco, the less likely it is that he or she will be able to quit later.[1] Access to tobacco is conducive to developing and maintaining a tobacco addiction. In field trials around the country, minors' attempts to purchase tobacco from stores and vending machines have been successful more than 50% of the time.[2–10] A survey of 10th graders in two Minnesota communities found a wide diversity of locations from which tobacco had been purchased.[11] In the United States, 947 million packs of cigarettes worth $1.23 billion are sold illegally to minors each year, and the tobacco industry derives 3% of its profits ($221 million) from such sales.[12]

Although most states have laws regulating sales to minors, these laws are rarely enforced.[5,7,13–16] An analysis of 11 active state and local enforcement efforts and 1 200 interviews with health experts, students, parents, and vendors from 18 states found that although 44 states and the District of Columbia have laws prohibiting the sale of tobacco to minors, only 32 violations in 5 states were documented.[13]

Previously, we reported the 6-month effects of a comprehensive merchant and community education program to reduce cigarette sales to minors.[2] One-year follow-up data are reported here.

Methods

In January 1988, 412 diverse stores in Santa Clara County, California that sold cigarettes over the counter and 30 outlets that had cigarette vending machines were visited by minors aged 14 to 16 years. In July 1988, after several months of intervention, which included comprehensive community education, direct merchant education, and contact with chief executive officers of chains and franchises, all stores were revisited by minors. Although the interventions concluded in July 1988, we wanted to assess their sustained effects. The primary data reported here were collected in January 1989, 1 year after the posttest and more than 6 months after the intervention ended. At this follow-up, a random sample of 25% of stores that sold tobacco over the counter, stratified by city (n = 7) and type of store (chain, franchise, independent), was selected and those that were still in business were visited by minors (n = 97). Because a new state law enacted in January 1989 made it illegal for minors to purchase tobacco, we obtained letters from local law enforcement officials allowing teenaged boys and girls to conduct this follow-up without being arrested.

Results

Statistically significant reductions from pretest to follow-up were generally

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This paper was submitted to the journal August 3, 1990, and accepted with revisions November 30, 1990.

TABLE 1—Cigarette Sales to Minors

	Pretest (all stores)	Pretest (stores with pretest and 1-year data)	6-Month Posttest (all stores)	1-Year Follow-up	P Value	% Change Pretest to 1-Year Follow-up and 95% CI[a,b]
All stores	74% (412)[c]	76% (97)	39% (408)	59% (97)	.01	−18% (−4%, −31%)
Chain stores	62% (174)	70% (40)	27% (174)	58% (40)	NS[d]	−13% (11%, −36%)
Franchise stores	81% (110)	82% (28)	50% (110)	50% (28)	.02	−32% (−5%, −59%)
Independent stores	85% (121)	79% (29)	47% (121)	69% (29)	NS	−10% (10%, −31%)
Gas stations	84% (50)	91% (11)	55% (50)	55% (11)	.10	−36% (7%, −80%)
Liquor stores	83% (103)	87% (23)	46% (103)	65% (23)	.06	−22% (1%, −44%)
Convenience stores	81% (103)	82% (28)	48% (103)	54% (28)	.03	−29% (−2%, −55%)
Pharmacies	60% (67)	53% (15)	28% (67)	53% (15)	NS	0% (−37%, 37%)
Grocery stores	60% (89)	65% (20)	22% (89)	65% (20)	NS	0% (−34%, 34%)
Sale to male minor	71% (180)	80% (70)	35% (87)	53% (70)	<.001	−27% (−11%, −43%)
Sale to female minor	77% (122)	65% (26)	47% (73)	77% (26)	NS	12% (−16%, 39%)
Warning sign posted	3% (13)	1% (97)	23% (92)	25% (97)	<.001	24% (14%, 34%)
Minor asked age	24% (100)	21% (96)	48% (196)	34% (96)	.05	14% (0%, 27%)

[a] % change equal to % in pre-test minus % in 1-year follow-up (% rounded).
[b] 95% confidence interval was calculated for change by using sample with data at pretest and 1-year follow-up.
[c] The number of stores for each variable is provided in parentheses.
[d] NS indicates a significance level greater than .10.

obtained even though sales increased between posttest and follow-up. Table 1 presents pretest, 6-month posttest, and 1-year follow-up data. Pretest data are provided for the entire sample of stores and for the sample of stores visited at both pretest and 1-year follow-up. The McNemar nonparametric test for the significance of changes and confidence intervals were calculated on the difference in percent change between pretest and 1-year follow-up for stores with both pretest and 1-year follow-up data.[17]

Discussion

There are discrepancies between what boys and girls are taught in school-based prevention programs and what they observe and experience outside of the classroom. In the classroom, they are told not to smoke, yet in the community, adolescents observe an abundant supply of tobacco, a willingness on the part of merchants to sell them tobacco, a lack of enforcement of laws regulating sales to minors, and very little evidence that society views the problem as a priority. Changing this discrepancy should be a public health priority.

The data reported here illustrate that a comprehensive merchant and community education campaign can achieve sustained effects in reducing sales to minors. However, without continued intervention, monitoring, and we now believe, sanctions, recidivism will occur. Because we wanted to test explicitly the effectiveness of a purely educational intervention, we did not encourage law enforcement to cite stores that sold tobacco to minors. Follow-up data reported here as well as data from other projects that utilized the sanctions of enforcement convince us of the importance of combining education of merchants and the public with the bite of enforcement.[18,19] The experience of the Solano County Cancer Prevention Program in California[19] suggests, that communities will respond more favorably to punitive enforcement efforts if educational interventions are implemented first. Once nonpunitive educational interventions relying on merchants' voluntary compliance are implemented and illegal sales are not reduced entirely, enforcement becomes a more acceptable option. Preceding enforcement with education also allows merchants an opportunity to change their business practices without the immediate threat of punishment.

It is reasonable to hypothesize that if minors have a difficult time obtaining tobacco, then they may be prevented from experimenting with and later becoming addicted to it. Along these lines, studies of school smoking policies have found that making smoking inconvenient may reduce tobacco use by adolescents.[20–22] Without doubt, there will always be some tobacco use by adolescents, just as there is illegal use of marijuana, crack, and other hard drugs. But the more risk young people must take to obtain tobacco and the more societal norms reject tobacco use by young people, the greater the likelihood that young people will not use tobacco. It is also reasonable to ask whether interventions to reduce tobacco sales to adolescents will actually reduce access or simply change where access occurs. Unless sales to adolescents are totally stopped, which is unrealistic in most communities, adolescents who really want tobacco will be able to get it. If, however, community norms and merchant practices about sales to adolescents are applied consistently and comprehensively, we predict that access will truly be reduced. As this study and others have shown, educating the community, merchants who sell tobacco, public policymakers, and law enforcement officials can contribute to reducing the sale of tobacco products to minors.

The 1990s will undoubtedly be a landmark decade for eliminating minors' access to tobacco now that communities around the country have begun addressing the problem. For example, in 1990, research in Minnesota resulted in national attention when dozens of communities throughout the state passed ordinances restricting minors' access to vending machines (Jean Forster, University of Minnesota, personal communication, 1990). The US government also has spoken out on the problem. In May 1990, HHS Secretary Sullivan recommended model state legislation to control minors' access.[14,15] These recommendations dealt with retail licensing, minimum age, graduated penalties for illegal sales, warning signs, enforcement, and vending machines.

Our research suggests that although long-term effects of comprehensive educational programs to reduce tobacco sales to minors can be achieved, interventions must be sustained and rely on multiple intervention strategies. Future intervention research should address minors' access to smokeless tobacco, challenges of intervening in densely populated urban areas, the effectiveness of education combined with enforcement and policy change, the sustainability of intervention effects, and the relationship between reducing tobacco access and youth tobacco use. ☐

Acknowledgments

This project was cosponsored by Stop Teenage Addiction to Tobacco (STAT) and the Santa Clara County Chapters of the American Cancer Society, American Heart Association, and American Lung Association. It was supported in part by grants to STAT from the Henry J. Kaiser Family Foundation, Menlo Park, Calif, the Cancer Research Foundation of America, Alexandria, Va, El Camino Hospital, Mountain View, Calif, Kiwanis and Rotary Clubs of Northern California, and members of STAT.

References

1. Johnston LD, O'Malley PM, Bachman JG. *National Trends in Drug Use and Related Factors among American High School Students and Young Adults, 1975–1986.* Washington, DC: National Institute on Drug Abuse; 1987.
2. Altman, DG, Foster, V, Fasenick-Douss, L, Tye, JB. Reducing the illegal sale of cigarettes to minors. JAMA 1989; 261:80–83.
3. Anonymous: Minors earily buy tobacco. DOC News and Views 1988; Spring:10.
4. DeClaire J. Babes in tobacco land: Can tougher laws snuff out cigarette sales to kids? View 1990;32:25.
5. DiFranza JR, Norwood BD, Garner DW, Tye JB. Legislative efforts to protect children from tobacco. JAMA 1987; 257:3387–3389.
6. Hoppock, KC, Houston, TP. Availability of tobacco products to minors. J Fam Pract 1990; 30:174–176.
7. Kirn TF: Laws ban minors' tobacco purchases, but enforcement is another matter. JAMA 1987; 257:3323–3324.
8. Naidoo J, Platts C. Smoking prevention in Bristol: getting maximum results using minimum resources. Health Educ J 1985; 44:39–42.
9. Skretny MT, Cummings KM, Sciandra R, Marshall J. An intervention to reduce the sale of cigarettes to minors. NY State J Med 1990; February:54–55.
10. Stanwick RS, Fish DG, Manfreda J, Gelskey D, Skuba A. Where Manitoba children obtain their cigarettes. Can Med Assoc J 1987; 137:405–408.
11. Forster JL, Kunt-Inge K, Jeffery RW. Sources of cigarettes for tenth graders in two Minnesota cities. Health Educ Res 1989; 4:45–50.
12. DiFranza JR, Tye JB. Who profits from tobacco sales to children. JAMA 1990; 263:2784–2787.
13. Office of the Inspector General. Youth access to cigarettes. May 1990; OEI-02-90-02310.
14. Office on Smoking and Health. State laws restricting minors' access to tobacco. MMWR 1990; 39:350–353.
15. Sullivan LW. Statement of Louis W. Sullivan, M.D., Secretary of Health and Human Services, before the Committee on Finance, U.S. Senate, May 24, 1990.
16. Uzych L. Some effective steps against teen-age tobacco use. American Medical News 1989; January 27:24.
17. Agresti A. *Categorical Data Analysis.* New York: Wiley; 1990.
18. Woodridge Police Department: *Report on Cigarette Sales to Minors.* Woodridge, IL; 1989.
19. Feighery E, Shaffer G, Descheemaeker N. Community education and enforcement efforts to reduce illegal tobacco sales to minors: A Case Study. Unpublished manuscript.
20. Porter A. Disciplinary attitudes and cigarette smoking: a comparison of two schools. Br Med J 1982;285:1725–1726.
21. Crow CS. Smoking areas on school grounds: are we encouraging teenagers to smoke? J of Adolesc Health Care 1984; 5:117–119.
22. Reid D. Prevention of smoking among school children: recommendations for a policy development. Health Educ J 1985; 44:3–12.

Hormone–PMS Relationship More Complex Than Previously Thought

Contrary to popular belief, a new study suggests that premenstrual syndrome (PMS) is not simply related to changes in the levels of reproductive hormones that occur during the premenstrual phase of a woman's cycle. Instead, PMS may be triggered by reproductive hormonal events, as yet unidentified, occurring before the premenstrual phase, or it may be the result of a cyclic mood disorder that is synchronized with (but not caused by) the menstrual cycle.

An estimated 5% of American women are affected by PMS and experience behavioral, emotional, and physical symptoms severe enough to impair their social or occupational functioning. Symptoms include sadness, irritability, anger, tension, loss of energy, and changes in appetite and sleeping patterns.

The study entitled "Lack of Effect of Induced Menses on Symptoms in Women with Premenstrual Syndrome," which appeared in the April 25 issue of the *New England Journal of Medicine*, was conducted by researchers at the National Institute of Mental Health (NIMH) and the National Institute of Child Health and Human Development, in Bethesda, Maryland. NIMH scientists Peter J. Schmidt, MD, and David R. Rubinow, MD, headed the investigation.

The study helps explain why hormone therapy attempting to correct a presumed abnormality during the premenstrual phase is not effective in treating PMS. The findings are consistent with results from previous studies showing the inefficacy of therapy with the hormone progesterone.

"This important study dramatically alters our understanding of and treatment approaches for PMS. It provides valuable new information about the disorder and will stimulate further research to help the millions of women suffering from PMS," said Alan I. Leshner, PhD, NIMH acting director. "Scientifically, the fact that the hormone–PMS relationship is not as simple as previously believed makes PMS a useful model for understanding the very complex relationship between biology and behavior."

What Scientists Funded by the Tobacco Industry Believe About the Hazards of Cigarette Smoking

K. Michael Cummings, PhD, MPH, Russell Sciandra, MA, Amy Gingrass, and Ronald Davis, MD

ABSTRACT

Despite overwhelming evidence documenting the hazards of cigarette smoking, the tobacco industry denies that smoking has been proven to cause disease. The industry professes a desire to clear up the smoking and health "question" and often points to its support of the Council for Tobacco Research (CTR) as evidence of its interest in investigating the health dangers of smoking. This paper presents results of a survey of CTR-funded scientists regarding their beliefs about the health dangers posed by smoking cigarettes. The vast majority of scientists funded by the CTR believe that cigarette smoking is an addiction that causes a wide range of serious, often fatal, diseases. This result suggests that the tobacco industry is unwilling to accept even the opinions of scientists it has deemed worthy of funding. Scientists should consider the ethical implications of accepting funds from the CTR and other tobacco industry-supported institutions. (*Am J Public Health*. 1991;81:894–896)

Introduction

The US Surgeon General has stated that "smoking represents the most extensively documented cause of disease ever investigated in the history of biomedical research."[1] Despite overwhelming scientific evidence against cigarettes, the tobacco industry continues to assert that controversy, debate, and uncertainty exist among scientists concerning smoking as an important cause of illness.[2–4]

In 1972, a confidential memorandum from a Tobacco Institute (TI) vice-president described TI policy as "creating doubt about the health charge without denying it, advocating the public's right to smoke without actually urging them to take up the practice, and encouraging objective scientific research as the only way to resolve the question of the health hazard."[5] Industry spokespersons often point to the industry's support of the Council for Tobacco Research (CTR) as evidence of corporate interest in obtaining scientific evidence on the "alleged" relationship between tobacco use and disease.[3]

The CTR, formed in 1954 by cigarette manufacturers, describes its primary mission as support of research into questions of tobacco use and health.[6] The council awards peer-reviewed research grants to independent scientists who are assured complete scientific freedom in conducting and publishing their studies. Since 1954, the council has provided more than $150 million for 1,108 original studies by more than 700 scientists.[6] In 1989, the CTR listed 204 active projects.

We present results of a survey of CTR-funded investigators, which characterizes what the investigators believe regarding the health effects of tobacco.

Methods and Materials

The study population included the principal investigators of research projects funded by the CTR in 1989.[6] Of 204 investigators listed, 179 were located at universities or institutions in the United States. This survey was restricted to those working in the United States.

Eligible survey participants were mailed a one-page questionnaire assessing their beliefs about the relation between cigarette smoking and various health complications and asking them to rank the importance of 10 different areas of tobacco research. Respondents were asked about their current and past research on tobacco and their cigarette smoking status. A cover letter stated that we were surveying "scientists who had published studies on smoking and health and/or have received research support from organizations interested in the tobacco and health issue."

Of 179 questionnaires mailed to eligible scientists in July 1990, 13 were returned with an incorrect mailing address. A total of 77 completed questionnaires were returned, which represents a response rate of 46% (77/166). No further attempt was made to elicit response. A comparison of responders and nonresponders revealed no significant differences in academic credentials (PhD vs MD), institutional affiliation (university vs other), or the nature of the CTR-funded project (i.e., a tobacco study vs a nontobacco study).

Results

Respondents were asked to "indicate the degree to which you believe the scientific evidence suggests a causal relationship with cigarette smoking" for

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eight separate health complications. With the exception of bladder cancer, nearly all respondents rated the relationship between smoking and illness as "strong" or "moderate" (Table 1). Only 1 of 77 respondents was a current smoker. Nationally, approximately 29% of adults smoke.[7]

Only 22% of respondents indicated that any of their current or past research focused on the health effects of tobacco use.

Ninety-four percent of respondents agreed with the statement "smoke from someone else's cigarette is harmful to a nonsmoker"; 91% agreed that most deaths from lung cancer are caused by smoking; and 76 of 77 agreed with the statement "cigarette smoking is addictive" (Table 2).

Rankings of the importance of 10 areas of research on tobacco varied widely (Table 3). Overall, research on preventing tobacco use received the highest ranking, followed by smoking cessation methods.

TABLE 1—Beliefs About the Scientific Evidence Suggesting a Causal Relationship Between Cigarette Smoking and Various Illness Conditions

	Strength of Causal Relationship with Cigarette Smoking:			
Illness/Condition	Strong %	Moderate %	Slight %	Not Established %
Emphysema	88.9	9.7	-	1.4
Chronic bronchitis	77.1	20.0	1.4	1.4
Heart disease	67.1	27.4	2.7	2.7
Low birth weight	51.6	30.6	12.9	4.8
Lung cancer	93.2	5.4	1.4	—
Cancer of the mouth, throat	76.1	19.4	1.5	3.0
Bladder cancer	24.6	56.1	15.8	28.1
Shorter life expectancy	76.4	16.7	4.2	2.8

TABLE 2—Beliefs About the Dangers of Environmental Tobacco Smoke, Smoking as a Primary Cause of Lung Cancer, and Smoking as an Addiction

Statement	Agree Strongly %	Agree Somewhat %	Disagree Somewhat %	Disagree Strongly %
The smoke from someone else's cigarette is harmful to a nonsmoker	52.9	41.4	5.7	—
Most deaths from lung cancer are caused by cigarette smoking	66.2	25.4	8.5	—
Cigarette smoking is addictive	83.3	15.3	1.4	—

TABLE 3—Mean Rankings of the Importance of 10 Areas of Research on Tobacco

Research Area	Mean Rank*	Standard Deviation	Percent Ranking Area as Top Priority
Tobacco use prevention	5.4	2.9	19%
Smoking cessation methods	5.2	2.8	14%
Neoplastic diseases	5.1	3.1	21%
Cardiovascular diseases	5.1	2.4	8%
Pregnancy and infant health	4.9	2.3	7%
Nonneoplastic respiratory diseases	4.6	2.3	—
Passive smoking	4.5	2.7	9%
Chemistry, pharmacology, toxicology	3.5	2.8	6%
Tobacco economics and legislation	3.4	3.4	12%
Tobacco advertising	3.4	2.9	4%

*Ranks range from low = 1 to high = 10.

Discussion

The low response rate is not unusual for a mailed questionnaire survey,[8] but does potentially limit the generalizability of the findings. The comparison of respondent and nonrespondent characteristics does not suggest any systematic response bias. It is possible that among those not responding to the survey were some who feared retribution from the tobacco industry. Such fear, if it existed, would likely be among those whose views are congruent with the majority of the respondents. It is also possible that those with less scientifically acceptable positions or greater commitment to the tobacco industry were less likely to respond, though it would seem to be in the industry's interest to have its views represented in such a survey, if possible. For these reasons, we believe the results accurately represent views of scientists funded by the CTR.

The survey shows that most scientists funded by the CTR believe cigarette smoking is an addiction that causes a wide range of serious, often fatal, diseases. This finding conflicts with the tobacco industry's description of the scientific community as divided on the question and indicates the industry does not accept the opinions even of scientists whose research it funds. Although acknowledging a need for additional investigation of the mechanisms linking smoking and disease, respondents gave the highest priority to research on tobacco use prevention and cessation. None of CTR's active research projects relate to these topics. Despite its stated mission to fund research into the etiology of diseases "alleged" to be related to tobacco use, only 1 in 6 CTR-funded scientists reported conducting research focused on the health effects of tobacco.

We suggest that rather than sponsoring a genuine "program of research into questions of tobacco use and health"[6] the CTR is a public relations vehicle intended to foster a false impression that cigarette manufacturers are interested in investigating the smoking and health "question." We believe such misuse of science raises serious ethical questions for scientists who accept funding through CTR or similar industry-supported entities. Even assuming that adequate funding is not available elsewhere, tobacco industry-supported scientists must ask themselves whether the value of their research in expanding the body of biomedical knowledge outweighs its utility in furthering the corporate interests of a business which kills 434,000 Americans every year.[9] □

References

1. *The Health Benefits of Smoking Cessation.* U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. DHHS Publication No. (CDC) 90-8416, 1990.
2. Resnik R. Where we stand. *Philip Morris Magazine.* September/October 1990; 39.
3. Warner KE. *Selling smoke: cigarette advertising and public health.* Washington, DC: American Public Health Association; 1986.
4. RJR ordered by FTC to show cause in Mr. FIT case. *Mealey's litigation reports: Tobacco.* April 1989; 4(4):7–8.
5. Cowley G. Science and the cigarette. *Newsweek.* April 1, 1988: 66–67.
6. *1989 Report of the Council for Tobacco Research—U.S.A., Inc.* New York: The Council for Tobacco Research—USA Inc; 1990.
7. *Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General.* U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, DHHS Publication No. (CDC) 89-8411, January 1989.
8. Dillman DA. *Mail and Telephone Surveys: The Design Method.* New York: John Wiley & Sons Inc; 1978.
9. Schultz JM. Smoking-attributable mortality and years of potential life lost—United States, 1988. *MMWR.* 1991; 40(4):62–71.

Gun-Related Deaths Increase Further for Black and White Young Men

The Department of Health and Human Services (HHS) reported today that one out of five deaths of teens and young adults in 1988 was gun related. Among young Black males, close to half of all deaths were firearm related.

Researchers at HHS' National Center for Health Statistics said the firearm-related death rate among Black and White males aged 15 to 24 years had declined in the early 1980s, but then rapidly increased from 1984 to 1988, especially for teenagers and particularly among Black males. Between 1984 and 1988, the firearm death rate among teenagers increased by over 40%, rising 20% from 1987 to 1988 alone, and reaching its highest level to date, 17.7 deaths per 100,000.

"This study shows that for the first time, the firearm death rates for both White and Black male teenagers exceeded the mortality from all natural causes of death," said HHS Secretary Louis W. Sullivan, MD. "For young Black males in particular, the excessive firearm and homicide death rates are appalling and heart-rending.

For Black males aged 15 to 19 years, the firearm death rate and firearm homicide rate more than doubled from 1984 to 1988, with an increase of 38% from 1987 to 1988 alone. By contrast, the nonfirearm homicide rate remained relatively stable. Black male teenagers were almost three times as likely to die from gun-related deaths than from all natural causes of death. For White males 15 to 19 years, the firearm death rate in 1988 for the first time exceeded that of natural causes, by 11%.

HHS Assistant Secretary for Health James O. Mason, MD, who heads the US Public Health Service, said, "The statistics show an American epidemic—without parallel in any other industrialized nation of the world. Physicians and other citizens need to work together to devise the same kinds of educational and prevention techniques that we use to attack other epidemics."

The findings are contained in a study, "Firearm Mortality Among Children, Youth and Young Adults, 1979–1988," produced by NCHS, a part of the Centers for Disease Control. The study examines homicide, suicide, and unintentional firearm deaths among those aged 1 to 34 years.

Does Cigarette Smoking Have an Independent Effect on Coronary Heart Disease Incidence in the Elderly?

Richard Benfante, PhD, MPH, Dwayne Reed, MD, PhD, and John Frank, MD, MSc

ABSTRACT

In order to evaluate the effects of cigarette smoking on coronary heart disease (CHD) in elderly persons in the Honolulu Heart Program, 1,394 men between ages 65 and 74 were followed during an average 12-year period for new cases of nonfatal myocardial infarction and fatal CHD. Incidence rates increased progressively in individuals classified at baseline as never, former, and current smokers, respectively. The absolute excess risk associated with cigarette smoking was nearly twice as high in elderly compared with middle-aged men. (*Am J Public Health*. 1991;81:897–899)

Introduction

While cigarette smoking is well established as a major risk factor for coronary heart disease (CHD) in middle-aged populations,[1–3] the question of whether it is a risk factor for CHD in the elderly has recently arisen because some studies have reported a diminished effect of cigarette smoking on CHD in older persons.[4–9] This reported lack of effect of cigarette smoking on CHD in the elderly is puzzling because the deleterious effects of smoking on CHD risk have been implicated at various stages of atherosclerosis, a pathologic process that generally progresses with age.[9–12] We had an opportunity to examine this question in an elderly group of the Honolulu Heart Program (HHP) cohort. The present analysis was undertaken to determine whether cigarette smoking behavior in the elderly predicts subsequent disease to the same degree as in middle-aged men.

Methods

In the HHP cohort, 1,394 men between ages 65 and 74 and free of clinical cardiovascular disease were followed for 12 years after baseline examination for new cases of definite CHD (non-fatal myocardial infarction and fatal CHD). Details of the differential diagnosis of CHD, surveillance procedures, and case ascertainment in the HHP have been previously described.[13–16]

In order to examine the effect of smoking on CHD risk, age-adjusted incidence rates were calculated by smoking status (current, former, and never) at baseline and expressed in terms of person-years of follow-up. The independent role of cigarette smoking on CHD risk was evaluated using a Cox proportional hazards model that included other major risk factors for CHD.[17,18] Relative risks for cigarette smoking were based on the risk ratio between current smokers and never smokers.

In order to see whether the effect of cigarette smoking on CHD risk was different in elderly compared to middle-aged men, 3,429 of the youngest men in the cohort (ages 51–59 at the examination) were studied as well.

Results

Average values for selected smoking characteristics in elderly and middle-aged men are presented in Table 1. Current elderly smokers on average smoked 20 cigarettes per day compared to 24 cigarettes per day for middle-aged men. Current elderly smokers smoked an average of 10 years longer than middle-aged smokers. For both elderly and middle-aged men, former smokers had higher daily consumption rates than current smokers.

In both age groups there was a consistent and progressive increase in CHD incidence rates by increasing level of smoking experience (Figure 1). Never smokers showed the lowest rates of CHD, followed by former smokers, with current smokers exhibiting the highest. When the relative risks for elderly and middle-aged men were compared in a Cox multivariate lifetable regression that included other major risk factors for CHD (systolic blood pressure, serum cholesterol, diabetes history, body mass index, and alcohol intake) the relative risk for the elderly was 1.62 (95% CI = 1.01, 2.61) compared to 1.80 (95% CI = 1.25, 2.61) for the younger men (Table 2). The difference in relative risks (relative risk ratio) between elderly and middle-aged men was not statistically significant. When an age-smoking interaction term was included in the model, it was also not statistically significant. From the perspective of excess risk

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TABLE 1—Average Values for Smoking Characteristics in Elderly and Middle-Aged Smokers at Baseline Examination

Smoking Characteristics	Smoking Status by Age Group at Examination			
	Ages 51–59		Ages 65–74	
	Past N = 1167	Current N = 1303	Past N = 527	Current N = 406
Cigarettes/day	27.5	23.8	26.3	19.7
Years Smoked	24.3	36.1	31.2	46.5
Years Quit	11.6	—	14.9	—

TABLE 2—Relative Risks* for Coronary Heart Disease in Elderly and Middle-Aged Men Based on Current Smoking Status at Baseline Examination

Age Group	N	b	S.E.	RR†	95% CI
65–74	1394	0.485	0.243	1.62	1.01, 2.61
51–59	3429	0.589	0.188	1.80	1.25, 2.61

*Based on Cox multivariate regression (includes age, cigarette smoking, systolic blood pressure, diabetes history, serum cholesterol, body mass index and alcohol intake).

†Determined by taking the exponent of the b-coefficient for smoking (Current—Never) in multivariate setting.

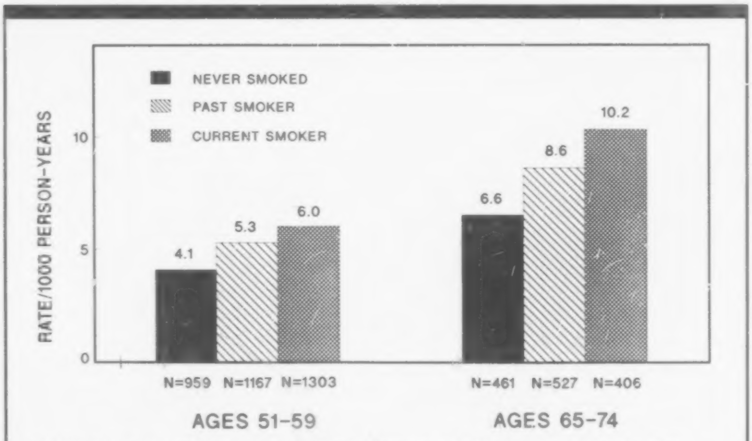

FIGURE 1—CHD Incidence by Smoking Status in Middle-Aged and Elderly Men

for CHD, the difference between current and never smokers for the elderly was 3.6 events per 1,000 person-years, compared to 1.9 events for middle-aged smokers.

Discussion

The results of the present study show that cigarette smoking continues to be an independent predictor of CHD incidence in this cohort of Japanese-American men over age 65, and that the effect is relatively undiminished compared with middle-aged men. CHD incidence for past smokers was intermediate between current and never smokers in both age groups. This is consistent with other evidence that the beneficial effects of quitting are effective later in life as well as during middle years.[1,19-21] It is also important to note that while the relative risk for cigarette smoking on CHD was similar in elderly and middle-aged men, there was nearly a two-fold increase in the excess absolute risk in the elderly men. Thus, since CHD rates increase exponentially with age, the public health impact of a risk factor such as smoking may be greater in older persons even though the relative risk is unchanged.

While a few other studies of older persons have reported an association of cigarette smoking with cardiovascular disease,[19-22] some prospective cohort studies report a diminished effect in the elderly.[4-9] Most notable is the Framingham study which has recently reported that the relationship between cigarette smoking and CHD disappears in both men and women over age 65.[7,8] Several factors may account for differences between the results of Framingham and Honolulu. One is the different analytical methods of relating risk factors to CHD. We used a single baseline measure with an average 12 years of follow-up, compared to the use of the most recent measure from a series of biennial examinations in Framingham. If there was a change in risk factor level, the most recent measure would be used; for example, if a smoker quit, he would be coded as a nonsmoker and then followed for subsequent disease.

Another factor is the potential intervention effect of numerous repeat examinations in Framingham versus just a few examinations in Honolulu. Subjects and their physicians could be notified of high risk levels numerous times in Framingham, but only a few times in Honolulu. Thus, behavioral change or medical intervention would be more likely to occur in Framingham.

There is also the selective attrition effects related to the different time periods of the two studies. The first 20 years of the Framingham follow-up were 1948–68, a period when CHD rates were the highest known in the US. The Honolulu study started in 1965 during a period of declining CHD mortality rates.[23,24] It is possible that these secular effects resulted in differences in the proportion of susceptible subjects reaching the elderly group in the two studies. In addition, the sample of men between ages 65–74 in Honolulu was about twice that for the same age group for men in Framingham, providing a distinct advantage in statistical power.

In conclusion, the present study supports the hypothesis that the biological or clinical effects of such a strong risk factor as cigarette smoking in middle age are maintained over time with increased exposure, and that the benefits of quitting accrue regardless of age. It is therefore recommended that the cessation of cigarette smoking be promoted as part of an overall strategy for prevention of CHD at all ages. ☐

Acknowledgments

This research was supported by National Heart, Lung, and Blood Institute contract NO1-HC-02901. The authors are grateful for the excellent programming assistance of Darryl Chiu.

References

1. US Department of Health, Education, and Welfare: Smoking and Health: A Report of the Surgeon General. DHEW Pub. No. (PHS) 79-50066. Washington, DC: Government Printing Office, 1979.
2. US Department of Health and Human Services: The Health Consequences of Smoking: Cardiovascular Disease: A Report of the Surgeon General. Pub. No. DHHS (PHS) 84-50204. Washington, DC: Government Printing Office, 1983.
3. Wilhelmsen L: Coronary heart disease: Epidemiology of smoking and intervention studies of smoking. Am Heart J 1988; 115:242–249.
4. Miettinen OS, Neff RK, Hershel J: Cigarette smoking and nonfatal myocardial infarction: Rate ratio in relation to age, sex and predisposing conditions. Am J Epidemiol 1976; 103:30–36.
5. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR: Predicting coronary heart disease in middle-aged and older persons. The Framingham Study. JAMA 1977; 238:497–499.
6. Stokes J, Kannel WB, Wolf PA, Cupples LA, D'Agostino RB: The relative importance of selected risk factors for various manifestations of cardiovascular disease among men and women from 35 to 64 years old: 30 years of follow-up in the Framingham study. Circulation 1987; 75(Suppl V):V65–V73.
7. Harris T, Cook EF, Kannel WB, Goldman L: Proportional hazards analysis of risk factors for coronary heart disease in individuals aged 65 or older. The Framingham Heart Study. J Am Geriatr Soc 1988; 36:1023–1028.
8. Castelli WP, Wilson WF, Levy D, Anderson K: Cardiovascular risk factors in the elderly. Am J Cardiol 1989; 63:12H–19H.
9. Hennekens C, Buring J, Mayrent S: Smoking and aging in coronary heart disease. *In:* Bosse R, Rose C (eds): Smoking and Aging. Lexington, MA: Lexington Books, 1984.
10. McGill HC: The cardiovascular pathology of smoking. Am Heart J 1988; 115:250–257.
11. FitzGerald GA, Oates JA, Nowak J: Cigarette smoking and hemostatic function. Am Heart J 1988; 115:267–271.
12. Mjos OD: Lipid effects of smoking. Am Heart J 1988; 115:272–275.
13. Worth RM, Kagan A: Ascertainment of men of Japanese ancestry in Hawaii through World War II Selective Service Registration. J Chronic Dis 1970; 23:389–397.
14. Belsky JL, Kagan A, Syme SL: Epidemiologic Studies of Coronary Heart Disease and Stroke in Japanese Men Living in Japan, Hawaii, and California. Hiroshima, Japan: Research Plan Atomic Bomb Casualty Commission, 1971; Technical Report 12–71.
15. Nichaman MZ, Hamilton HB, Kagan A, Grier T, Sacks ST, Syme SL: Epidemiologic studies of coronary heart disease and stroke in Japanese men living in Japan, Hawaii and California: Distribution of biochemical risk factors. Am J Epidemiol 1975; 102:491–501.
16. Yano K, Reed DM, McGee DL: Ten-year incidence of coronary heart disease in the Honolulu Heart Program. Am J Epidemiol 1984; 119:653–666.
17. Cox DR: Regression models and life tables. J R Stat Soc B 1972; 34:187–220.
18. Kalbleisch JD, Prentice RL: The Statistical Analysis of Failure Time Data. New York: John Wiley and Sons, 1980; Ch 4.
19. Jajich CL, Ostfeld AM, Freeman DH Jr: Smoking and coronary heart disease mortality in the elderly. JAMA 1984;252:2831–2834.
20. Hermanson B, Omenn GS, Kronmal RA, Gersh BJ: Beneficial six-year outcome of smoking cessation in older men and women with coronary heart disease: Results from the CASS registry. N Engl J Med 1988; 319:1365–1369.
21. Weintraub WS, Lloyd KW, Seelaus PA, Agarwal JB, Helfant RH: Importance of total life consumption of cigarettes as a risk factor for coronary heart disease. Am J Cardiol 1985; 55:669–672.
22. Siegel D, Kuller L, Lazarus NB, Black D, Feigal D, Hughes G, Schoenberger JA, Hulley SB: Predictors of cardiovascular events and mortality in the systolic hypertension in the elderly program pilot project. Am J Epidemiol 1987; 126:385–399.
23. Stallones RA: The rise and fall of ischemic heart disease. Sci Am 1980; 243:53–59.
24. Thom TJ, Epstein FH, Feldman JJ, Leaverton PE: Trends in total mortality and mortality from heart disease in 26 countries from 1950 to 1978. Int J Epidemiol 1985; 14:510–520.

Staff Involvement and Special Follow-up Time Increase Physicians' Counseling About Smoking Cessation: A Controlled Trial

Carol Duncan, RD, MPH, Morton J. Stein, MD, and Steven R. Cummings, MD

ABSTRACT

We compared the counseling behaviors of two groups of health maintenance organization physicians: one group received training about smoking cessation counseling; the other group received the same training plus staff support and appointment time specially designated for follow-up of smokers. We interviewed patients after their office visits to measure smoking counseling. The group receiving staff support and designated follow-up time counseled more and made more follow-up appointments about smoking. (*Am J Public Health.* 1991;81:899–901)

Introduction

Only a minority of smokers recall being counseled to quit by physicians[1,2] and internists who do counsel smokers seldom use effective strategies such as setting quit dates and making follow-up appointments.[3,4] We have found that even after intensive training in counseling, physicians discussed smoking with only half of their smoking patients and infrequently made follow-up appointments about smoking cessation.[5,6] To determine whether greater involvement of administrative and office staff, and provision of specified follow-up time, would increase physician counseling, we conducted a controlled trial.

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TABLE 1—Baseline Characteristics of Patients in Sample Groups

Characteristics	Training-Plus Before	Training-Plus After	Training-Only
Number of patients:	102	105	139
Female (%)	28	36	43
Mean age (years)	45	46	48
College graduates (%)	34	34	27
Mean cigarettes/day	18	16	17
Occasional smoker (%)	11	18	9
Want to quit smoking (mean, 10-pt. scale)	7.0	7.2	7.1
Confidence in quitting (mean, 10-pt scale)	5.0	5.9	5.2
First visit to this doctor (%)	4	28	27
Type of visit (%)			
Routine follow-up	44	53	50
Urgent problem	33	19	17
Check-up/physical	21	27	29
Other	2	1	4

Methods

We studied two physically separate medical stations at a Kaiser-Permanente Medical Center in the San Francisco Bay Area where internists had not been previously trained in smoking cessation and each saw a minimum of 20 patients per week. Four internists at one medical station agreed to receive training with nursing and administrative support (training-plus), while five internists at another station agreed to receive the training (training-only). One training-plus physician subspecialized in pulmonology; one training-only physician subspecialized in endocrinology, and another in infectious disease. One physician in the training-plus group currently smoked. Two physicians in each group were female, and the mean ages of the two groups were similar (36 years for training-plus; 34 years for training-only).

Research staff met with administrators, then with nurses and their supervisors, then with training-plus physicians and nurses to plan the system of administrative supports. Nurses agreed to ask patients if they smoked, to mark the charts of smokers, and to ask smokers to fill out smoking history forms. Physicians decided to use pre-stamped Nicorette prescription pads. The Department of Medicine designated two appointment slots per week per provider for follow-up of smokers. The group adopted a follow-up protocol using a two-part written Quit-date prescription: one copy was given to the patient and one copy was filed by quit date for nurses to use to telephone patients on quit dates.

All providers concurrently attended two one-hour seminars designed to demonstrate and practice counseling about smoking cessation. These seminars have been described in detail.[5] Training for the training-plus group differed in that nurses and their supervisors attended the sessions. Physicians in the control station were encouraged to ask their office staffs to use reminder stickers to identify smokers.

We defined a smoker as anyone who had smoked a tobacco cigarette during the past seven days. Research staff surveyed patients in the waiting areas, and asked all smokers to participate. This continued until a minimum of 15 smokers were enrolled per physician. Participants answered a questionnaire about their smoking habit, desire to quit and confidence in ability to quit. As soon as possible after the visit, an interviewer telephoned each smoker to determine if the physician or staff had discussed smoking with them and, if so, what steps the physician had recommended. These data were first collected before any meetings with the training-plus group. The week after training was completed data collection began simultaneously from both training-only and training-plus groups' patients.

To monitor use of designated follow-up appointments for smoking we tallied computerized visit records for "smoking" appointments. We also verified with physicians which of their appointments had actually been used for counseling smokers.

Since outcomes were measured by patient report, patients were the unit of analysis. Statistical significance was tested using t-test for continuous and chi-square for categorical data. We also used logistic regression to adjust for differences in outcomes between the groups, for baseline differences in demographic characteristics in patients, their confidence in quitting, occasional smoking status and type of visit. Because these adjustments did not affect results, we present unadjusted differences in the tables.

TABLE 2—Comparison of Physician Counseling About Smoking Between Trained Groups

–	Training-only	Training-plus	Difference	(95% C.I.)
(%)				
Smoking discussed	48.9	68.7	19.8	(6.5,33.2)
Physician initiated discussion of smoking	27.1	62.6	35.5	(22.5,48.6)
Physician asked for quit date	15.8	28.3	12.5	(0.8,24.2)
Smoker agreed to date	7.5	18.2	10.7	(1.0,20.4)
Smoker received written reminder of date	2.3	15.2	12.9	(4.5,21.3)
Nicotine gum prescribed	6.8	15.3	8.5	(−0.7,17.7)
Referred to program	10.5	25.5	15.0	(4.0,26.0)
Received self-help booklet	9.8	34.3	24.6	(13.1,36.1)
Given follow-up appointment about smoking	9.0	18.8	9.8	(−0.4,19.8)

*Smallest sample sizes: 131 for training-only and 96 for training-plus due to missing data.

Results

We enrolled 335 patients; 26 per physician both before and after training in the training-plus group, and 28 per physician in the training-only group. Telephone interviews were completed for 96 percent of patients within 72 hours of their visits. Patients in the three study groups had similar characteristics, but those enrolled during the pre-training period were less confident of being able to quit, were less often making first visits and more often had urgent problems than those enrolled during the post-training period (Table 1). Compared with smokers in the training-plus group, more patients in the training-only group were regular smokers.

Training-plus physicians discussed smoking with their smoking patients more often than training-only physicians, initiated the discussion more often, asked for more quit dates, gave more written reminders and booklets, referred more patients to groups, programs or professional help, prescribed nicotine gum more often, and made more follow-up appointments than training-only physicians (Table 2). Individual physicians in the training-plus group counseled from 52 to 93 percent of patients, while those in the training-only group counseled from 32 to 63 percent of patients. Office staffs of training-plus physicians discussed smoking with 5.2 percent of patient smokers while office staffs of training-only physicians discussed smoking with none.

Comparing their performance from before the program started to after they received training and office support, physicians in the training-plus group counseled more patients (from 38 to 69 percent), initiated the discussion more often (25 to 63 percent), suggested quit dates more often (3 to 28 percent), prescribed nicotine gum more often (4 to 15 percent), referred more patients to programs (11 to 26 percent), gave more booklets (12 to 34 percent), and made more follow-up appointments (10 to 19 percent). All differences were statistically significant ($P < .05$ for all comparisons).

Forty (60 percent) of 68 available smoking appointments were filled, and 23 (34 percent) of designated appointments were actually used for smokers. Only five (22 percent) of these 23 smokers failed to keep their appointments.

Discussion

In this pilot study we found that involvement of staff and administrators in planning and support of smoking cessation counseling enhanced the effect of physician training. Solberg and Kottke have suggested that it is important to enlist office staff in support of smoking cessation efforts in medical practices[7] and the National Cancer Institute has emphasized involvement of staff and administration in smoking cessation in its most recent booklet for health professionals. We found this approach to be quite acceptable to these volunteers, but it is rarely used in practice. For example, we have found that 60 percent of health maintenance organization physicians never have other staff counsel and 68 percent never make follow-up appointments about quitting.[3]

As expected, the official designation of time for follow-up visits substantially increased follow-up of smokers. Not all appointments were used for smokers, but the total number (23) may have been appropriate since physicians were advised to make appointments with smokers who agreed to quit dates, and 69 smokers had so agreed. There is concern that smokers may frequently fail to keep follow-up appointments about cessation, but the proportion of missed appointments by smokers was within the 15–33 percent range reported for all patients in adult medical practices.[8–10]

This was a pilot study. Our small number of physician subjects were volunteers who were not randomized, and telephone interviews were not done blindly. The end point was physician counseling, not smoking cessation. Larger and longer-term randomized trials are needed to determine whether organizational involvement and follow-up appointments increase rates of smoking cessation among patients as well as to identify which components are most effective.

We conclude that systematic staff support and designated follow-up visits for smokers increase smoking cessation efforts of physicians beyond those that result from training alone. Whether these interventions increase cessation rates deserves further study. □

Acknowledgments

This work was supported by grant #CA38374 from the National Cancer Institute. Dr. Cummings' work is supported in part by the Henry J. Kaiser Family Foundation Faculty Fellowship in General Internal Medicine.

References

1. Anda RF, Remington PL, Sienko DDG, Davis RM: Are physicians advising smokers to quit? The patient's perspective. JAMA 1987; 275:1916–1919.
2. Louis Harris and Associates: Health Maintenance-A nationwide survey of the barriers toward better health and ways of overcoming them, conducted among representative samples of the American public, business and labor leaders. Newport Beach, California: Pacific Mutual Life Insurance; 1978.
3. Cummings SR, Stein MJ, Hansen B, Richard RJ, Gerbert B, Coates TJ: Smoking counseling and preventive medicine. A survey of internists in private practice and a health maintenance organization. Arch Intern Med 1989; 149:345–349.
4. Ockene JK, Amy J, Goldberg R, *et al:* A survey of Massachusetts physicians' smoking intervention practices. Am J Prev Med 1988; 4:14–20.
5. Cummings SR, Coates TJ, Richard RJ, *et al:* Training physicians in counseling about smoking cessation. A randomized trial of the "Quit for Life" program. Ann Intern Med 1989; 110:640–647.
6. Cummings SR, Richard RJ, Duncan CL, *et al:* Training physicians about smoking cessation: A controlled trial in private practices. J Gen Intern Med, 1989; 4:482–489.
7. Solberg LI, Kottke TE: Doctors helping smokers. A new way to intervene. Minn Med 1988; 71(7):413–441.
8. Deyo RA, Inui TS: Dropouts and broken appointments. A literature review and agenda for future research. Med Care 1980; 18:1146–1157.
9. Starkenburg RJ, Rosner F, Crowley K: Missed appointments among patients new to a general medical clinic. NY State J Med 1988; 9:473–475.
10. Goldman L, Freidlin R, Cook EF, *et al:* A multivariate approach to the prediction of no-show behavior in a primary care center. Arch Intern Med 1982; 142:563.

Tobacco Consumption Declining Faster in Canada Than in the US

Murray J. Kaiserman, PhD, and Byron Rogers, BA

ABSTRACT

Consumption and prices of tobacco products in Canada and the United States are compared over the period 1981 to 1989. In Canada, consumption has fallen from a higher level and at a faster rate than in the United States (3.52 kg/adult to 2.48 kg/adult vs 3.21 kg/adult to 2.48 kg/adult). With respect to cost, the real tobacco price index in Canada has grown faster than its US counterpart. These results are attributed to higher taxation practices, vigorous legislation, and the leadership role adopted by the Canadian government during the 1980s. (*Am J Public Health.* 1991:81:902–904)

Can a vigorous national policy that includes high taxation and strong legislation towards smoking bring about significant declines in tobacco consumption? In Canada, the answer is yes.

The most recent figures on total tobacco product consumption expressed as kg of tobacco per person aged 15 years and older show that Canada's consumption fell from a level that was higher than that of the United States in 1981 to the same, lower level by 1989 (Table 1). The choice of the age of 15 years reflects the fact that adult smoking patterns are typically established between the ages of 15 and 18 years.[1,2]

In 1981, Canadians aged 15 or older, consumed 3.52 kg of tobacco per capita.[3] Their American cousins, on the other hand, consumed 3.21 kg per capita in 1981, or 8.8% less.[4]

By the end of 1989, Canadians, like Americans, consumed 2.48 kg annually. For the Canadians, this represented a decrease of 22.8% in total weight of tobacco products consumed. By the end of the decade, the Canadian per capita tobacco consumption rate had declined to reach the same level as its United States counterpart (Figure 1).

Over the 9-year period, the slope of the decline in per capita tobacco consumption in Canada has been about 30% steeper than the trend in the United States. Statistical analysis with a multiregression model shows that the difference in decline between Canada and the United States is statistically significant and is probably attributable to policies within the country rather than external influences.

One of the determinants in short-term tobacco demand is the real price of tobacco products, that is, the price adjusted for inflation.[4,5] Graphs of the real tobacco price index and tobacco consumption for both countries show the expected inverse relationship (Figures 2 and 3).

Canada's steeper tobacco price index curve is indicative of that country's greater willingness to tax tobacco, both federally and provincially (Figure 4). In Canada, federal and provincial taxes together account for approximately two thirds of the retail price of a pack of cigarettes. In the United States, federal and state taxes account for approximately 20% of the retail price.[1,4]

In the 1989 Canadian Federal Budget, the finance minister cited health reasons, in addition to fiscal considerations, in justifying a $0.35 (US) increase per pack of 20 in federal cigarette taxes. Provinces impose their own tobacco taxes in addition to the federal tax, thus further raising the real price of tobacco products. In Canada, the average federal and provincial tax rate is now about $2.25 (US) per pack of 20 whereas in the United States the average federal and state tax rate is $0.38 (US) per pack of 20.[4]*

In addition, the 1989 Canadian Federal Budget reduced the tax rate gap between fine-cut tobacco or roll-your-own (RYO) and manufactured cigarettes. Preliminary sales data reported to Statistics Canada indicate that, in the first half of 1990, consumption of RYO tobacco, long the choice of those Canadian smokers who preferred not to quit in response to cigarette price increases, is now also declining. Cigarette rolling tobacco accounts for 14% of the Canadian cigarette market, but only about 1% of the market in the United States.[1,4]

A factor in Canada's relatively recent trend toward higher tobacco tax rates has been the federal government's adoption of a comprehensive approach to discouraging tobacco use by Canadians. Key elements of the National Strategy to Reduce Tobacco Use include a ban on tobacco product advertising, restrictions on promotional activities, prominent new health warning

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This paper was submitted to the journal August 20, 1990, and accepted with revisions March 6, 1991.

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*On Feb. 26, 1991 the finance minister increased the federal tax by 52 cents per pack of 20. After subsequent provincial budgets, the average tax rate is now about $3.24 per pack of 20 or about 75% of the retail price.

messages on tobacco product packaging, health promotion and community support activities around smoking prevention and cessation, economic adjustment programs to assist tobacco growers leaving the industry, and bans or restrictions on smoking in workplaces and on common carriers within federal jurisdiction.

Internationally, Canada has taken a leadership role in banning tobacco advertising, increasing the prominence and legibility of health warning labels, and restricting smoking on longer-duration commercial airline flights.[6] Like Canada, the United States has now banned smoking on flights of up to 6 hours duration, and

TABLE 1—Real Tobacco Price Indexes and Per Capita Consumption of Tobacco Products Consumed in Canada and the United States Between 1981 and 1989

Year	Real Tobacco Price Index in Canada[5]	Per Capita Consumption in Canada	Real Tobacco Price Index in United States[4]	Per Capita Consumption in United States[4]
1981	100.0	3.52	100.0	3.21
1982	104.1	3.51	104.8	3.17
1983	115.7	3.32	121.4	3.06
1984	123.6	3.19	124.2	2.92
1985	139.5	3.08	127.0	2.91
1986	157.4	2.90	133.1	2.80
1987	163.7	2.78	137.6	2.68
1988	173.0	2.69	144.2	2.62
1989	199.3	2.48	155.1	2.48

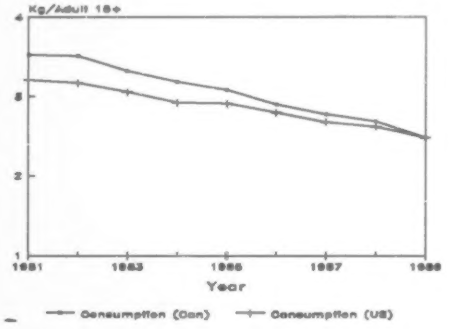

FIGURE 1—Comparison of per capita tobacco consumption in the United States and Canada (1981–1989)

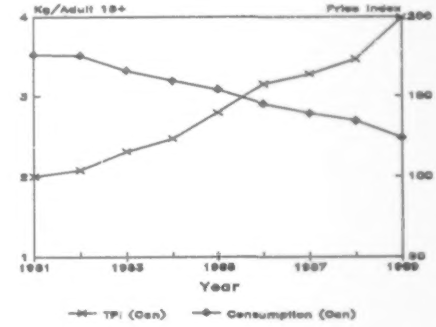

FIGURE 2—Tobacco consumption and tobacco prices in Canada (1981–1989)

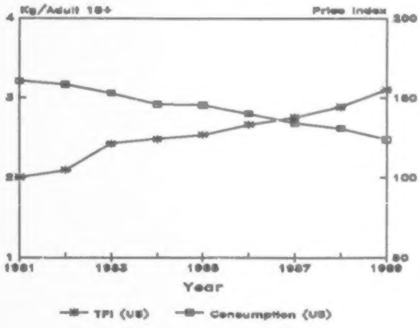

FIGURE 3—Tobacco consumption and tobacco prices in the United States (1981–1989)

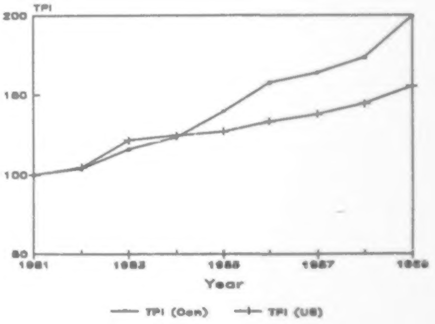

FIGURE 4—Comparison of real tobacco price indices in the United States and Canada (1981–1989)

the US Congress is considering proposals to further restrict or ban cigarette advertising.

For policymakers, the conclusions are simple. Although education and smoking cessation programs can result in moderate declines in tobacco consumption, major declines can be achieved through increased taxation and advertising bans. As a result of an across-the-board strategy to reduce tobacco use, Canada, whose citizens were once characterized as "champion smokers," has caught up with and appears poised to overtake the United States in the trend toward butting out. □

References

1. US Department of Health and Human Services, Public Health Service. *Reducing the Health Consequences of Smoking: 25 Years of Progress. A Report of the Surgeon General.* Washington, DC: Govt Printing Office; 1989. DHHS publication (CDC) 89-8411.
2. New Zealand Toxic Substances Board. *Health or Tobacco: An End to Tobacco Advertising and Promotion.* Wellington, New Zealand: Department of Health; May 1989.
3. Statistics Canada. *Production and Disposition of Tobacco Products.* Statistics Canada; monthly, December 1989. Catalogue No. 32-022.
4. US Department of Agriculture, Economic Research Services. *Tobacco Situation and Outlook Report.* Washington, DC: United States Department of Agriculture; April, 1990. Economic Research Services Catalogue TS-206.
5. Statistics Canada. *Consumer Prices and Price Indexes.* Statistics Canada; quarterly, 1981–1990. Catalogue No. 62-010.
6. Labour Canada: Non-smokers' Health Regulations, amendment. *Canada Gazette.* 1990; 124(13):2428 (Part II) SOR/DORS/90-335.

The Impact of Time in Treatment on the Employment and Earnings of Drug Abusers

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ABSTRACT

We use data from a longitudinal survey to estimate the effects of time in drug abuse treatment on post-treatment weeks worked and earnings for 2,420 clients in three treatment modalities. The regression analysis shows that time in treatment had a positive and statistically significant impact on these labor market outcomes, but the effects were small for all modalities. Although residential clients experienced the largest relative changes in weeks worked and real earnings, a benefit-cost calculation suggests that additional residential treatment cannot be justified from earnings improvements alone. These results may indicate a need for more employment services while in treatment. (*Am J Public Health.* 1991; 81:904–907)

Introduction

Although recent studies have shown that drug abuse treatment is successful in lowering drug use and criminal activity for some clients,[1–6] little is known about the impact of treatment on clients' employment and earnings.[7] This paper uses multivariate regression models to examine the impact of time in treatment on post-treatment weeks worked and real (inflation-adjusted) earnings of over 2,400 drug abusers.

Methods

We use data from the Treatment Outcome Prospective Study (TOPS), a long-term, large-scale study of 11,000 drug abusers who entered treatment in 1979, 1980, and 1981 at 41 selected drug abuse treatment programs.[4] It provides extensive information about the nature of drug abuse treatment and describes characteristics and behavior of drug abusers prior to treatment, at admission, and at several follow-up intervals. The sample for this study includes all 2,420 clients who participated in the 12-month follow-up interview—the largest follow-up cohort in TOPS. Hubbard and colleagues[4] report and compare sociodemographics at intake and follow-up from a TOPS program for clients in each modality. Rather than repeating those descriptive statistics here, we refer the reader to their book.

We develop regression models to understand the variation in employment and earnings outcomes. For example, consider a simple regression model that describes a labor market outcome as a function of several explanatory variables[8,9]:

$$Y_f = X_f\beta + \gamma PRETOPS + \delta TOPS + \phi POSTTOPS + \varepsilon, \qquad (1)$$

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[*] Because methadone maintenance usually involves long-term treatment, some individuals were still in a TOPS treatment program at the time of their follow-up interview. For these clients, the period "one year following discharge" actually refers to the period starting one year following admission.

TABLE 1—Regression Coefficient Estimates for the "Real Earnings" Specification by Treatment Modality[a]

Variables	Modality Methadone Maintenance (N = 737)	 Residential (N = 655)	 Outpatient Drug-Free (N = 750)
Intercept	$-14591.00^{\dagger\dagger}$	-2445.50	$-11573.00^{\dagger\dagger}$
	(2460.93)	(1637.52)	(2217.65)
Males	$2918.51^{\dagger\dagger}$	$1679.90^{\dagger\dagger}$	$4113.92^{\dagger\dagger}$
	(633.08)	(534.57)	(651.68)
Caucasian/white	$3351.42^{\dagger\dagger}$	$2034.48^{\dagger\dagger}$	$1953.35^{\dagger\dagger}$
	(679.22)	(516.64)	(780.67)
Highest grade attended	$965.71^{\dagger\dagger}$	$247.74^{\dagger\dagger}$	$1133.72^{\dagger\dagger}$
	(129.48)	(102.59)	(138.97)
Age (months)	2.78	3.36	0.78
	(3.52)	(2.69)	(3.76)
Married	$1209.12^{\dagger\dagger}$	736.65	602.12
	(590.49)	(537.15)	(677.57)
Head of household	$1463.22^{\dagger\dagger}$	-77.72	$2112.84^{\dagger\dagger}$
	(604.54)	(456.72)	(645.49)
Number of dependents	$707.14^{\dagger\dagger}$	137.17	$970.03^{\dagger\dagger}$
	(216.64)	(203.18)	(267.93)
Total weeks in treatment prior to entering a TOPS treatment program	-1.47	-2.20	$-14.21^{\dagger}$
	(3.62)	(4.29)	(7.83)
Total weeks in a TOPS treatment program	$21.09^{\dagger\dagger}$	$43.90^{\dagger\dagger}$	14.31
	(9.54)	(8.27)	(14.58)
Total weeks in treatment 12 months after leaving a TOPS treatment program	$-29.80^{\dagger\dagger}$	-18.35	-31.12
	(13.46)	(14.67)	(23.45)
Admitted to a TOPS program in:			
Chicago	912.02	-1190.83	—
	(1125.91)	(893.80)	
New Orleans	$4626.42^{\dagger\dagger}$	-694.88	-1433.59
	(1250.65)	(941.13)	(1465.23)
New York	1316.49	-341.41	$-2214.14^{\dagger}$
	(1007.28)	(1171.37)	(1292.49)
Phoenix	$2263.77^{\dagger\dagger}$	$-2025.50^{\dagger\dagger}$	-1146.80
	(1150.48)	(1016.83)	(1107.85)
Portland	—	-1155.11	-1186.32
		(965.59)	(1167.57)
Miami	1708.15	550.36	58.88
	(1313.09)	(948.43)	(1298.62)
San Francisco	--	-385.33	$-5519.56^{\dagger\dagger}$
		(982.57)	(1553.10)
R-square	0.21	0.13	0.22

[a] Dependent variable is total real earnings (1983 dollars) from full- or part-time jobs during the 12 months after leaving a TOPS program.

†Significantly different from zero at the 90% confidence level (two-tailed test).

††Significantly different from zero at the 95% confidence level (two-tailed test).

Note: Standard error in parentheses.

where Y_f is a labor market outcome variable at the one-year follow-up, X_f is a vector of explanatory variables at the one-year follow-up, *PRETOPS* is a measure of total time in treatment prior to entering a TOPS program, *TOPS* is total time in a TOPS program, and *POSTTOPS* is total time in treatment during the year after leaving a TOPS program.* From Equation (1) we can estimate the effect of TOPS treatment on a labor market outcome variable, controlling for demographics and prior and ensuing treatment episodes.

Equation (1) may yield biased parameter estimates for the treatment duration variables if unobserved client differences (e.g. motivation, discipline, aggression) are correlated with the time in TOPS treatment.[10] To demonstrate this situation conceptually, consider Equations (2) and (3), which explain labor market variable Y at admission to a TOPS program (Y_a) and one year after leaving a TOPS program (Y_f), respectively:

$$Y_a = X_a\beta + \gamma PRETOPS + \upsilon + \varepsilon_a \qquad (2)$$

$$Y_f = X_f\beta + \gamma PRETOPS + \delta TOPS + \phi POSTTOPS + \upsilon + \varepsilon_f \qquad (3)$$

where υ is an error term. If υ includes unobserved client characteristics, single-equation least-squares regressions may yield biased coefficient estimates. To correct this potential problem, we propose a "first-difference" model as illustrated in Equation (4):

$$(Y_f - Y_a) = (X_f - X_a)\beta + \delta TOPS + \phi POSTTOPS + (\varepsilon_f - \varepsilon_a). \qquad (4)$$

By differencing the variables at follow-up with the same variables at admission, we eliminate the error component, υ, and all other variables that do not change between admission and follow-up. The treatment duration coefficient, δ, now represents an unbiased estimate of the effect of time in a TOPS treatment program on labor market changes.

Results

Table 1 reports regression estimates for the real earnings specification of Equation (1). Coefficient estimates for the time in TOPS treatment variable were positive for each modality and statistically significant for methadone maintenance and residential clients. On average, one additional week in treatment increased annual real earnings for methadone clients by $21 and for residential clients by $44 during the year following TOPS treatment.

The dependent variable in the Table 2 equations is total weeks worked during the 12 months after leaving a TOPS program. Coefficient estimates for time in a TOPS treatment program were positive and statistically significant for every modality. The coefficients for the residential and outpatient drug free equations imply that six to seven additional weeks in treatment led to one additional week of employment. Methadone clients had a much lower estimated return, with 16 additional weeks in treatment needed for each additional week of employment.

In addition to demographic and treatment duration variables, Tables 1 and 2 report coefficient estimates for program location. Differences in city location coefficients reflect geographical differences in labor markets and program-specific differences in treatment delivery.

Table 3 lists coefficient estimates for the first-difference specifications. Several variables "difference out" because they did not change from admission to follow-up (e.g. race, sex, weeks in treatment prior to TOPS). Focusing on the time in treatment variables, we see that total

TABLE 2—Regression Coefficient Estimates for the "Weeks Worked" Specification by Treatment Modality[a]

	Modality		
Variables	Methadone Maintenance (N = 753)	Residential (N = 667)	Outpatient Drug-Free (N = 766)
Intercept	−24.273[††]	10.645[††]	4.635
	(6.066)	(5.394)	(4.994)
Males	7.012[††]	5.243[††]	7.119[††]
	(1.563)	(1.755)	(1.470)
Caucasian/white	6.522[††]	5.663[††]	6.156[††]
	(1.681)	(1.698)	(1.749)
Highest grade attended	2.165[††]	0.815[††]	1.859[††]
	(0.321)	(0.337)	(0.315)
Age (months)	−0.002	−0.002	−0.021[††]
	(0.009)	(0.009)	(0.008)
Married	1.390	1.235	1.433
	(1.459)	(1.767)	(1.538)
Head of household	3.733[††]	−2.268	3.046[††]
	(1.490)	(1.501)	(1.461)
Number of dependents	1.365[††]	0.792	1.389[††]
	(0.532)	(0.674)	(0.609)
Total weeks in treatment prior to entering a TOPS treatment program	−0.015 (0.009)	0.003 (0.014)	−0.043[††] (0.018)
Total weeks in a TOPS treatment program	0.061[††]	0.168[††]	0.137[††]
	(0.023)	(0.027)	(0.033)
Total weeks in treatment 12 months after leaving a TOPS treatment program	−0.054 (0.033)	−0.032 (0.049)	−0.032 (0.053)
Admitted to a TOPS program in:			
Chicago	0.155	−13.963[††]	—
	(2.781)	(2.957)	
New Orleans	7.231[††]	−5.280[†]	−4.[illegible]9
	(3.077)	(3.107)	(3.304)
New York	0.564	−9.432[††]	−10.379[††]
	(2.479)	(3.874)	(2.920)
Phoenix	8.899[††]	−10.995[††]	−4.106
	(2.846)	(3.348)	(2.522)
Portland	—	−11.661[††]	−6.624[††]
		(3.224)	(2.639)
Miami	6.008[†]	−4.696	−0.344
	(3.213)	(3.131)	(2.947)
San Francisco	—	−10.829[††]	−20.119[††]
		(3.260)	(3.539)
R-squared	0.184	0.162	0.213

[a] Dependent variable is total weeks worked at full- or part-time jobs during the 12 months after leaving a TOPS program.

[†] Significantly different from zero at the 90% confidence level (two-tailed test).

[††] Significantly different from zero at the 95% confidence level (two-tailed test).

Note: Standard error in parentheses.

weeks in a TOPS treatment program had a positive impact on real earnings and weeks worked in all equations. Only the coefficient estimate from the real earnings specification for methadone clients was not significantly different from zero.

Discussion

This is the first longitudinal study to analyze the impact of length of stay in drug abuse treatment on labor market outcomes. Regression analyses indicate that time in treatment had a statistically significant positive, but small, impact on the follow-up employment and earnings of clients in the major treatment modalities. We believe this result can be explained, in part, by the dearth of training and employment services that were available to treatment clients during this period. Treatment resources were often directed at addiction control and behavior modification at the expense of vocational services. To reverse this trend, greater investments in vocational and employment services may be necessary to produce a strong and lasting effect on labor market outcomes.

Interestingly, time in treatment prior to and following TOPS treatment had a generally negative, but insignificant, effect on real earnings and weeks worked for each modality. This result implies that individuals with more treatment prior to and following TOPS (holding constant time in TOPS treatment) are less likely to have labor market success. Perhaps treatment episodes before and after TOPS signal chronic treatment and employment problems for these clients.

Overall, residential clients experienced the largest impact from time in treatment. From the regression results, we found that residential clients would have earned approximately $44 more if they stayed an additional week in treatment. But the average weekly cost of residential treatment for each TOPS client was approximately $143.[4] Looking only at the earnings outcome of time in treatment, the investment in additional residential treatment would not be cost-beneficial. However, after factoring in the dollar benefits of other treatment outcomes (e.g. lower drug use, reduced criminal activity, improved health status), residential treatment is likely to generate a higher return on the investment. Further analyses of the treatment process, treatment outcomes, and linkages between criminal activity, drug use, and employment are necessary to help design contemporary drug abuse treatment strategies. ☐

Acknowledgments

This work was supported by Grant R01 DA05599-01 from the National Institute on Drug Abuse, Public Health Service, US Department of Health and Human Services. The views expressed in this paper are those of the authors and do not represent an official position of the National Institute on Drug Abuse or the US Department of Health and Human Services.

We gratefully acknowledge the programming support of Anne Theisen, MA. We are also indebted to Maria Bachteal, Sharon Barrell, Judy King, and Judy Parsons for their assistance in preparing tables and editing the manuscript.

References

1. Anglin MD, Hser Y: Treatment of drug abuse. *In:* Wilson JQ, Tony M (eds): Drugs and Crime, University of Chicago Press, Chicago, 1990.
2. Ball JC, Lang WR, Myers CP, Friedman SR: Reducing the risk of AIDS through methadone maintenance treatment. J Health Soc Behav 1988; 29:214–226.
3. Harwood HJ, Hubbard RL, Collins JJ, Rachal JV: The costs of crime and the benefits of drug abuse treatment: A cost-benefit analysis using TOPS data. *In:* Leukefeld CG, Tims FM (eds): Compulsory Treatment of Drug Abuse: Research and Clinical Practice. Research Monograph Series 86. Rockville, MD: National Institute on Drug Abuse, 1988.
4. Hubbard RL, Marsden ME, Rachal JV, Harwood HJ, Cavanaugh ER, Ginzburg

TABLE 3—Regression Coefficient Estimates for the "First-Difference" Specifications by Treatment Modality

	Dependent Variable – Difference in Real Earnings[a]			Dependent Variable – Difference in Weeks Worked[b]		
Variables	Methadone Maintenance (N = 707)	Residential (N = 626)	Outpatient Drug-Free (N = 714)	Methadone Maintenance (N = 732)	Residential (N = 646)	Outpatient Drug-Free (N = 766)
Intercept	−1477.69	−1634.75††	−200.37	−4.90††	−2.31	−1.29
	(1028.17)	(693.39)	(585.78)	(2.25)	(1.79)	(1.56)
Difference in highest grade attended	731.19††	−3.29	131.28	0.79	0.33	−0.47
	(283.45)	(179.77)	(204.17)	(0.63)	(0.46)	(0.53)
Difference in age	−1.71	26.92	21.04	0.01	0.08	0.06
	(30.77)	(20.74)	(16.39)	(0.07)	(0.05)	(0.04)
Difference in marital status	−247.21	209.12	1851.95††	0.29	0.53	6.13††
	(696.24)	(724.34)	(674.66)	(1.54)	(1.85)	([illegible]5)
Difference in household status	1036.43†	−1057.83††	675.97††	4.75††	−3.91††	2.93††
	(643.02)	(537.78)	(511.40)	(1.42)	(1.38)	(1.33)
Difference in number of dependents	435.32	275.70	490.32†	2.19††	0.62	1.09
	(276.75)	(277.17)	(272.80)	(0.61)	(0.72)	(0.74)
Weeks in a TOPS treatment program	10.43	34.60††	24.45†	0.06††	0.14††	0.07††
	(12.98)	(12.68)	(13.56)	(0.03)	(0.03)	(0.04)
Weeks in treatment year after TOPS	3.86	−8.11	−23.61	−0.04	0.02	−0.04
	(17.69)	(22.26)	(21.12)	(0.04)	(0.06)	(0.06)
R-square	0.02	0.03	0.03	0.05	0.05	0.04

[a] Dependent variable is total real earnings during the year after leaving a TOPS program minus total real earnings during the year prior to entering a TOPS program.

[b] Dependent variable is total weeks worked during the year after leaving a TOPS program minus total weeks worked during the year prior to entering a TOPS program.

†Significantly different from zero at the 90% confidence level (two-tailed test).

††Significantly different from zero at the 95% confidence level (two-tailed test).

Note: Standard error in parentheses.

HM: Drug Abuse Treatment: A National Study of Effectiveness. Chapel Hill, NC: University of North Carolina Press, 1989.

5. McAuliffe WE, Ch'ien JMN, Launer E, Friedman R, Feldman B: The Harvard group aftercare program: Preliminary evaluation results and implementation issues. *In:* Ashery RS (ed): Progress in the Development of Cost-effective Treatment for Drug Abusers, NIDA Research Monograph 58. Rockville, MD: National Institute on Drug Abuse, 1985.
6. McLellan AT, Woody GE, Luborsky L, O'Brien CP, Druley KA: Increased effectiveness of substance abuse treatment: A prospective study of patient-treatment 'matching': J Nerv Ment Dis 1983; 171:597–605.
7. French MT, Rachal JV, Harwood HJ, Hubbard RL: Does drug abuse treatment affect employment and earnings of clients? Benefits Q 1990; 1:58–67.
8. Luft HS: The impact of poor health on earnings. Rev Econ Stat 1975; 57:43–57.
9. Mincer J: Schooling, experience, and earnings. New York: National Bureau of Economic Research, 1975.
10. Chamberlain G: Analysis of covariance with qualitative data. Rev Econ Studies 1980; 47:225–238.

Television Viewing and Obesity in Adult Females

ABSTRACT

We measured the relation between time spent watching television per week and obesity in 4,771 adult females. After controlling for age, education, cigarette smoking, length of work week, and weekly duration of exercise, females who reported three to four hours of TV viewing per day showed almost twice the prevalence of obesity (body fat > 30 percent), and those who reported more than four hours of TV watching per day showed more than double the prevalence of obesity, compared to the reference group (<1 hr/day). Part of the TV/obesity association was a function of differences in exercise duration among the four TV viewing categories. (*Am J Public Health.* 1991;81:908–911)

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Introduction

Lifestyle plays a key role in the development and prevention of a host of chronic diseases. Until recently, few have recognized television viewing as a significant lifestyle factor. Yet, next to sleep and work, watching TV consumes more time than any other activity in America.[1,2] Adolescents spend more time watching TV each year than they spend in school, and the typical adult averages nearly four hours of TV viewing per day.[1–3]

Research has determined that frequent television viewing may function as a risk factor in a number of disorders.[4–10] Health problems may result from many hours of daily TV watching due to the deceptive and inaccurate health-related messages which are regularly transmitted to viewers.[10,11] However, television's principal problem is probably more related to the physical passivity which accompanies TV viewing rather than the misleading micro-lessons it teaches.

TV has profoundly changed the use of leisure time in America.[2,12] While people watch TV, physical activity tends to be minimal and snacking is prevalent.[10,13,14] Conditions for hypokinetic ailments such as obesity and poor fitness dominate.

In 1986, Tucker[6] studied the connection between television viewing and multiple measures of physical fitness in adolescent males. Findings showed that high levels of TV watching were strongly associated with low scores on tests of fitness, (pullups, situps, sidestep, six-minute run, and pushups). Similarly, Dietz and Gortmaker[5] found that as TV viewing increased, obesity increased systematically in several thousand children aged 6–11 and 12–17. In 1989, Tucker and Friedman[8] showed that time spent watching TV was directly related to obesity levels in adult males. Men who watched more than three hours of TV per day had more than twice the prevalence of obesity compared to those who watched less than one hour per day. To date, the association between TV viewing and obesity in adult females has not been studied; hence, the present investigation was conducted. A secondary purpose was to ascertain the extent to which age, cigarette smoking, hours worked per week, and weekly exercise duration mediate the relation between TV viewing and obesity in women.

Methods

Subjects

Subjects were 4,771 females employed by over 30 different companies in the United States. All subjects worked for companies which paid to have their employees participate in a health promotion/disease prevention screening program offered by Health Advancement Services, Inc. Participants were distributed nearly equally across family income categories ranging from $10,000 to $60,000+. Median age of the sample was 35 years and approximately 83 percent of the females were White, 60 percent were married, 18 percent separated or divorced, and 50 percent reported some college education.

Instrumentation and Procedures

All data were gathered by registered nurses employed by Health Advancement Services as part of their ongoing screening program. Subjects completed an informed consent form and each was tested individually during a private 50-minute examination. A structured, written questionnaire was used to collect the demographic and lifestyle information, including TV viewing, exercise, and smoking habits. A Harpenden skinfold caliper was employed to assess subcutaneous fat at three body sites on each subject: ***thigh***, vertical fold taken on the anterior aspect midway between the hip and the knee; ***triceps***, vertical fold taken on the back of the arm midway between the shoulder and the elbow; *iliac crest*, diagonal fold taken 2.5 cm above the iliac crest. The sum of the skinfold measurements along with age were used to estimate the total body fat percentage of each subject.[15] Research shows that this three-site skinfold method of

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TABLE 1—Hours of Television Viewing, Body Fat, and Obesity According to the Control Variables

Control Variables	N	Hours of TV per Day (row %)				Body Fat Percentage (row %)			
		<1	1–2	3–4	>4	<18	18–23	24–29	30+*
Total Group	4,771	12.3	59.2	24.1	4.4	10.7	26.2	30.9	32.3
Age (years)									
19–29	1,541	10.4	56.5	26.8	6.3	20.9	36.5	25.8	16.8
30–39	1,350	13.5	62.3	21.2	3.0	8.9	29.4	32.7	29.0
40–49	997	17.0	60.1	20.3	2.6	3.1	18.6	35.5	42.8
50–59	654	9.3	62.5	25.5	2.7	2.4	12.4	30.9	54.3
60+	229	5.3	50.2	34.2	10.2	2.3	7.7	33.5	56.6
Highest Education									
elementary	71	11.6	37.7	37.7	13.0	6.1	21.2	25.7	47.0
high school	1,941	8.5	57.3	28.6	5.6	11.6	25.2	30.7	32.6
vocational	357	10.7	55.2	29.6	4.5	7.4	24.3	33.1	35.1
college	1,726	14.6	61.1	20.5	3.9	12.3	29.5	29.9	28.3
professional	676	18.3	63.8	16.4	1.5	6.5	22.5	33.4	37.6
Smoking Status									
never smoked	2,451	13.1	60.1	23.3	3.5	9.9	25.2	29.4	35.6
ex-smoker	881	13.5	60.2	22.5	3.9	8.3	24.5	32.5	34.7
1–20 daily	1,020	9.9	56.2	27.7	6.2	15.3	29.4	32.2	23.1
21+ daily	419	9.3	58.9	27.9	4.0	10.5	31.0	31.5	26.9
Weekly Exercise									
<30 minutes	2,735	10.5	57.5	26.7	5.2	9.9	24.4	29.8	35.9
31–60 min	421	13.2	61.4	22.0	3.4	11.4	27.5	31.7	29.4
61–100 min	426	15.0	59.7	22.0	3.4	7.6	28.4	35.9	28.1
101–200 min	655	14.2	61.0	21.3	3.5	11.4	29.3	33.4	25.9
201+ min	534	16.4	63.0	17.7	2.9	16.2	29.8	29.2	24.8
Work Week									
<20 hrs/wk	215	8.0	43.4	32.6	16.0	8.1	24.4	36.4	31.1
21–40 hrs/wk	2,953	11.0	58.6	26.0	4.4	11.7	27.4	29.7	31.2
41–50 hrs/wk	1,422	14.7	63.0	19.9	2.4	9.7	24.6	32.0	33.7
51+ hrs/wk	181	19.6	60.3	18.4	1.7	6.2	26.4	30.9	36.5

*Females with 30 percent or more body fat were labeled obese.

body fat assessment correlates well with hydrostatic weighing (r = .84).[15] Obesity was defined as 30 percent or more body fat.[16] Body fat, television viewing, and the control variables were categorized as depicted in Table 1. Indirect validation of the body fat,[8,17–20] TV,[4,6–8,19] and exercise duration[17–20] variables has been demonstrated previously.

The relation between duration of TV viewing and body fat was indexed by the Mantel-Haenszel chi-square statistic which shows the extent of the linear association between ordinal-level, categorical variables.[21,22] The relation between TV viewing and obesity was measured by the odds ratio[23] with subjects who reported less than one hour of TV watching per day used as the reference group. Cochran-Mantel-Haenszel summary risk estimates were used to assess the TV/obesity relation with the potential confounders controlled.[24]

Results

Almost 60 percent of the subjects reported watching one to two hours of TV per day, while approximately one in four indicated three–four hours of daily TV viewing (see Table 1). More than four hours of TV viewing per day was reported most often by subjects who were young or old, by the uneducated, by smokers, by non-exercisers, and by those with short work weeks. Obesity, measured in nearly one-third of the adult females, was more common among older subjects, the uneducated, never and ex-smokers, non-exercisers, and those with long work weeks.

Table 2 shows the estimated risk of obesity by TV viewing time without adjustment and with adjustment for the control variables displayed in Table 1. After adjustment for all of the potentially confounding variables, subjects who reported four+ hours of daily TV watching showed more than two times the prevalence of obesity, and subjects who reported three–four hours of daily viewing showed almost twice the prevalence of obesity compared to females who watched less than one hour of TV daily (see Table 2).

Age had the strongest confounding effect on the TV/obesity relation of any single control variable. After adjustment for age only, estimated risk of obesity was increased by an average of 15 percent for the three TV viewing groups compared to the reference group. Control of exercise duration, in addition to the other potentially confounding factors, also affected the TV/obesity association. Specifically, estimated risk of obesity was decreased by approximately 18 percent for the four+ hour/day viewers and 12 percent for the three–four hour/day viewers compared to the reference group.

Discussion

According to the results, adult females who watch four or more hours of TV per day tend to have more than double the prevalence of obesity compared to those who watch less than one hour daily, as in adult males[8] and children.[5] However, because of the correlational nature of this study, cause-and-effect conclusions are not warranted. Although excessive TV viewing may actually contribute to obesity in women, it is plausible that obesity leads to an abundance of TV watching. As with adult males,[8] females who are fat may be

TABLE 2—Estimated Risk of Obesity by Television Viewing Group

		Obese*			
Daily TV Viewing	Variable Controlled	N	%	RR_{cmh}	95% CI
<1 hour (n = 585)	none	140	23.9	1.00*	—
1–2 hours (n = 2,825)	none	896	31.7	1.48	1.20, 1.81
	age, education, smoking, work week			1.46	1.15, 1.86
	above + exercise duration			1.29	0.99, 1.68
3–4 hours (n = 1,151)	none	402	34.9	1.71	1.36, 2.14
	age, education, smoking, work week			1.98	1.51, 2.61
	above + exercise duration			1.89	1.37, 2.61
>4 hours (n = 210)	none	73	34.8	1.69	1.20, 2.38
	age, education, smoking, work week			2.62	1.51, 4.53
	above + exercise duration			2.15	1.15, 4.01

Note: RR_{cmh} = Cochran-Mantel-Haenszel summary estimate of relative risk.
*Subjects who were found to have 30 percent or more body fat were labeled obese.

more inclined to watch TV as a primary source of recreation because of the minimal exertion required, whereas non-obese women may prefer other pastimes which demand greater physical involvement.

The strong association between TV watching and obesity in females could be the function of a "third variable" or confounding factor. Each of the control variables of the present study was strongly related to both TV viewing and obesity, except smoking, which was strongly associated with obesity only. Simultaneous adjustment for differences in age, education, length of work week, smoking, and weekly exercise duration substantially increased the estimated risk of obesity for the television watchers, especially the high-duration viewers.

Duration of weekly exercise had a meaningful impact on the TV/obesity relation. High-duration TV viewing was 80 percent more common and obesity was 45 percent more common among non-exercisers compared to those in the highest exercise group (see Table 1). Apparently, part of the reason women who watch a lot of TV tend to be more obese is because they tend to exercise less. If these women spent more time exercising, perhaps in place of some TV viewing time, risk of obesity would likely be decreased.

Other factors not measured in this investigation could confound the TV/obesity association. Diet is a good example. Because television watchers digest thousands of commercial and program messages which encourage consumption of non-nutritious foods,[25–30] and because snacking and eating foods which are advertised on TV are closely linked with television viewing,[10,13,14] it is possible that high-duration TV watchers are more obese than low-duration TV viewers because they consume more food, particularly more junk food, than their counterparts.

Perhaps the most cogent explanation of the association between TV viewing and obesity is based on a dynamic model: as TV viewing time increases, exercise tends to decrease and snacking tends to increase. As exercise decreases and snacking increases, obesity tends to increase. And as obesity increases, attraction to passive recreation, such as watching TV, tends to increase—a cycle of mutual interaction and reinforcement. Future research relative to the TV/obesity relation will need to consider the dietary intake of subjects as well as their physical activity levels. Furthermore, because subjects in this sample were mostly White and well-educated, generalization to other populations will require additional study.

Without question, watching television is a powerful and pervasive lifestyle factor in our society. Given the enormous amounts of time devoted to this idle pastime and the many subtle and distorted health-related messages conveyed by the medium, it is not surprising that children and adults are influenced by TV to a degree far surpassing earlier beliefs.[1–3] Greater effort needs to be directed toward the study of television's influence on health and its role as a precursor of disease. □

References

1. Comstock G, Chaffee S, Katzman N, McCombs M, Roberts D: Television and Human Behavior. New York: Columbia University Press, 1978.
2. Pearl D, Bouthilet L, Lazar J (eds): Television and Behavior: Ten Years of Scientific Progress and Implications for the Eighties (Vol. 2). Washington, DC: US Govt Printing Office, 1982.
3. Singer DG: A time to reexamine the role of television in our lives. Am Psychol 1983; 38:815–816.
4. Tucker LA: Television's role regarding alcohol use among teenagers. Adolescence 1985; 20:593–598.
5. Dietz WH, Gortmaker SL: Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. Pediatrics 1985; 75:807–812.
6. Tucker LA: The relationship of television viewing to physical fitness and obesity. Adolescence 1986; 21:797–806.
7. Tucker LA: Television, teenagers, and health. J Youth Adolesc 1987; 16:415–425.
8. Tucker LA, Friedman GM: Television viewing and obesity in adult males. Am J Public Health 1989; 79:515–518.
9. Singer JL, Singer DG: Psychologists look at television. Am Psychol 1983; 38:826–834.
10. Gerbner G, Morgan M, Signorielli N: Programming health portrayals: What viewers see, say, and do. *In:* Pearl D, Bouthilet L, Lazar J (eds): Television and Behavior: Ten Years of Scientific Progress and Implications for the Eighties (Vol. 2). Washington, DC: US Govt Printing Office, 1982.
11. Hamburg BA, Pierce CM: Television and health: Introductory comments. *In:* Pearl D, Bouthilet L, Lazar J (eds): Television and Behavior: Ten Years of Scientific Progress and Implications for the Eighties (Vol. 2). Washington, DC: US Govt Printing Office, 1982.
12. Comstock G: Television and American social institutions. *In:* Pearl D, Bouthilet L, Lazar J (eds): Television and Behavior: Ten Years of Scientific Progress and Implications for the Eighties (Vol. 2). Washington, DC: US Govt Printing Office, 1982.
13. Clancy-Hepburn K, Hickey A, Neville G: Children's behavior responses to TV food advertisements. J Nutr Educ 1974; 6:93–96.
14. Duesere S: The Effects of Television Advertising on Children's Eating Habits. Unpublished doctoral dissertation, University of Massachusetts, Amherst, 1976.
15. Pollock ML, Schmidt DH, Jackson AS: Measurement of cardiorespiratory fitness and body composition in the clinical setting. Comprehensive Ther 1980; 6:12–17.
16. Sharkey B: Physiology of Fitness. Champaign, IL: Human Kinetics, 1984.
17. Tucker LA, Friedman GM: Walking and serum cholesterol in adults. Am J Public Health 1990; 80:1111–1113.
18. Tucker LA, Aldana S, Friedman GM: Cardiovascular fitness and absenteeism in the workplace. Am J Health Promo 1990; 5:140–145.
19. Tucker LA: Television viewing and physical fitness in adults. Res Q Exerc Sport 1990; 61:315–320.
20. Tucker LA: Physical fitness and psychological distress. Int J Sport Psychol 1990; 21:185–201.
21. Mantel N: Chi-square tests with one degree

of freedom: Extensions of the Mantel-Haenszel procedure. J Am Stat Assoc 1963; 58:690–700.

22. Mantel N, Haenszel W: Statistical aspects of the analysis of data from retrospective studies of disease. JNCI 1959; 22:719–748.
23. Kelsey JL, Thompson WD, Evans AS: Methods in Observational Epidemiology. New York: Oxford University Press, 1986.
24. Landis RJ, Heyman ER, Koch GG: Average partial association in three-way contingency tables: A review and discussion of alternative tests. Int Stat Rev 1978; 46:237–254.
25. Mauro FJ, Feins RP: Kids, Food and Television: The Compelling Case for State Action. Report for the Office of Research and Analysis Program and Committee Staff. Albany: New York State Assembly, March 1977.
26. Masover L: Television Food Advertising: A Positive or Negative Contribution to Nutrition Education? Unpublished master's thesis, Northwestern University, Evanston, IL, 1977.
27. Barcus FE, McLaughlin L: Food Advertising on Children's Television: An Analysis of Appeals and Nutritional Content. Newtonville, MA: Action for Children's Television, 1978.
28. Choate RB: Statements before the Subcommittee on Communications of the Committee on Interstate and Foreign Commerce. Washington, DC: US House of Representatives, July 14, 1975.
29. Choate RB: Testimony before the Federal Trade Commission in the matter of a trade regulation rule on food nutrition advertising. Washington, DC: Council on Children, Media and Merchandising, 1976.
30. Kaufman L: Prime-time nutrition. J Communication 1980; 30:37–46.

Characteristics of a Random Sample of Emergency Food Program Users in New York: I. Food Pantries

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ABSTRACT

Food pantry users throughout New York State were studied and many demographic differences found between New York City and Upstate New York respondents. Seven percent of households had no income and median income as percent of the poverty level was 59 percent. Slightly more than 40 percent were spending over 60 percent of their incomes on housing. The data from this survey, the first in New York State to employ a random sampling design, demonstrate a sizable gap between household needs and available resources. (*Am J Public Health*. 1991;81:911–914)

Introduction

Food pantries and soup kitchens have become symbols of hunger and poverty in the United States. Food drives are an accepted feature of life and the Emergency Feeding System (EFS), once regarded as a response to a temporary situation, has become institutionalized. As this occurs, government agencies, scholars, hunger activists, and emergency food providers have begun to question the advisability of expanding or even maintaining the system. Many issues are involved in the emerging debate, including the needs of those using the system, which was the focus of our studies.

New York is one of a few states that provides funding for the operation of its EFS which is composed of food pantries, soup kitchens, and food banks. In 1987 there were 414 pantries in New York City (NYC) serving 2.9 million people and 1,143 in upstate New York plus Long Island (Upstate) serving 2.4 million.[1] This and the following report[2] present data from a survey of persons utilizing the food pantry and soup kitchen components of the EFS throughout the state.

Methods

The food pantry survey was conducted from October 1988 to February 1989, at a random sample of sites drawn from the Bureau of Nutrition census.[1] At each site, selection of individual clients was as random as conditions permitted. The final sample included 311 clients from 29 pantries Upstate and 208 clients from 19 sites in NYC.

The interviewers, mainly students in social science and nutrition, were trained at Syracuse University or Hunter College. The 20-minute interview included primarily closed-ended questions about client demographics, sources and amounts of income, and participation in food and other assistance programs.

The census had shown that the numbers of people served per pantry ranged from a few to 128,000 per year. To verify that our random sampling procedure had preserved this diversity, we found no significant difference between the percentages of clients coming from larger or smaller sites in either Upstate ($\chi^2 = 0.547$, df = 1, $p > 0.3$), or NYC ($\chi^2 = 0.040$, df = 1, $p > 0.8$).

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TABLE 1—Characteristics of Food Pantry Clients

	% of Responses			
	Upstate (n = 311)	New York City (n = 208)	χ^2 Value	p
Female respondent	67	61	1.847[a]	0.174
Age of respondent (years)				
35 or under	50	34		
36–59	37	37		
60 or older	13	29	24.100[b]	0.0001
Ethnicity				
White	57	16	85.575[a]	0.0001
African-American	31	65	60.702[a]	0.0001
Hispanic	11	17	3.911[a]	0.048
Living/employment situation				
Live alone	23	39	14.950[a]	0.0001
Household with children	61	35	32.077[a]	0.0001
Someone in household employed	29	22	3.535[a]	0.060

[a] χ^2 (df = 1).
[b] χ^2 (df = 2).

TABLE 2—Income Sources of Food Pantry Clients

	% of Households[b]			
	Upstate	New York City	χ^2 Value[a]	p
Wages	24	20	1.268	0.260
Other public assistance	22	13	7.096	0.008
Social Security	21	26	2.096	0.148
SSI/other disability	20	14	2.597	0.107
AFDC (Aid to Families with Dependent Children)	21	16	1.869	0.172
Child support/alimony	8	1	12.582	0.0001
Retirement/pension	6	2	3.909	0.048
Home relief	5	7	0.576	0.448
Veteran's benefits	3	3	0.046	0.831
Unemployment benefits	2	2	0.135	0.713

[a] χ^2 (df = 1).
[b] Percentages do not add up to 100% because more than one response was possible.

TABLE 3—Income Range and Relation to Poverty Level

	% of Responses			
Household Income[a] (Monthly)	Upstate (n = 293)	New York City (n = 188)	Test Statistic	p
\$0	7	8		
\$1–300	22	25		
\$301–600	35	45		
\$601–1000	24	16		
>\$1000	12	6	11.380[b]	0.023
Income < 50% of poverty	41	44	0.424[c]	0.515
Income < 100% of poverty	83	80	0.682[c]	0.409
Median % of poverty	59%	59%	−0.157[d]	0.875
Median monthly per capita income	\$162	\$177	1.255[d]	0.209

[a] Excludes food stamps.
[b] χ^2 (df = 4).
[c] χ^2 (df = 1).
[d] Wilcoxon Rank Sum Test.

Results

As shown in Table 1, more of the NYC sample was age 60 or older. Within the older age group, there was no difference between geographic areas in the percentage of persons living alone; however, among respondents ages 36–59, a larger percentage lived alone in NYC than Upstate.

Income sources are presented in Table 2. Approximately 50 percent of the Upstate sample received public assistance, while 36 percent of the NYC households did so. Household income distributions (Table 3) were significantly different due to a larger percentage of households with higher total incomes Upstate. Approximately 40 percent of each sample had incomes less than 50 percent of poverty.

Sixty-two and 52 percent of Upstate and NYC respondents, respectively, were on Medicaid (χ^2 = 5.030, df = 1, p = 0.025). Approximately one-third of each group received a housing subsidy. Despite this, 45 percent of Upstate and 41 percent of NYC households spent more than 60 percent of their incomes on housing (shelter and utilities).

There was a highly significant difference (χ^2 = 18.286, df = 3, p = 0.0001) in length of time that households had utilized food pantries due to both more long-term users (>3 years) Upstate and more short-term users (<6 months) in NYC. Table 4 shows dissimilarities in households' use of other food programs with regard to use of soup kitchens, school meal programs, and senior meals.

Table 5 presents selected demographic characteristics of food stamp users. In both groups, neither living alone or living with others, nor educational level were related to food stamp use. Upstate, significantly more females, more households with children, and fewer seniors were on the program. In NYC, only employment status was significantly different.

The monthly food stamp benefit per household was significantly different ($129 Upstate and $95 NYC) and the Upstate per capita benefit was also significantly higher. Both samples showed significant differences between food stamp users and non-users with regard to income as a percentage of poverty level (Table 6).

Discussion

Since federal poverty income thresholds are based upon the cost of a mini-

TABLE 4—Use of Other Food Programs by Food Pantry Clients

	% of Households			
	Upstate (n = 311)	New York City (n = 209)	χ^2 Value[a]	p
All households[b]				
Food stamps	57	50	1.875	0.171
Soup kitchens	19	40	25.157	0.0001
Free food	41	50	4.079	0.043
Households with people over 60	(n = 60)	(n = 72)		
Meals at senior center	5	32	15.022	0.0001
Home-delivered meals	5	7	0.263	0.608
Households with children under 18	(n = 191)	(n = 78)		
School breakfast (free/reduced)	36	54	5.573	0.018
School lunch (free/reduced)	63	72	1.628	0.202

[a] χ^2 (df = 1).
[b] Percentages do not add up to 100% because more than one response was possible.

mally adequate diet, households below the poverty level are, by definition, at risk of being unable to purchase such a diet.[3] The median income of all participating households was 59% of poverty. Furthermore, federal poverty guidelines were developed when shelter costs were far lower, and the large percentage of income now spent by households further reduces the amount available for food.

Some differences were noted between Upstate and NYC respondents with regard to the extent of use of food programs. People Upstate had utilized pantries for a longer period of time, and there was a positive significant relationship between food stamp participation and length of food pantry use. Food stamp users also made greater use of other food programs. These tendencies suggest that these households are dependent on the EFS even though they are making use of the welfare system in a fairly inclusive manner. In NYC there was not, in most cases, significantly greater use of other programs by food stamp recipients, although the actual percentages of those using soup kitchens, senior meals, etc, was often higher than in the Upstate sample.

Receipt of welfare or food program benefits does not preclude the need for emergency food. Nearly half of respondents were receiving public assistance, but in New York State in 1988 AFDC plus full food stamp benefits brought a household to only 89% of the poverty level.[4] In the Upstate and NYC samples, respectively, 38 percent and 40 percent of respondents 65 and over lived on only Social Security payments which places them near the poverty level.[5] Nearly one-fourth of households included a wage-earner, but a full time minimum wage job brought a household of three to 74 percent of poverty and a household of four to only 57 percent of poverty.[6]

The percentage of households having incomes below 50 percent of the poverty level is similar to the nationwide figure.[7] These households more frequently had children, and, not surprisingly, were less likely to be employed. In both locations, non-White households were significantly more likely to have an income below 50 percent of poverty.

Our findings portray a population in need, and at risk of the health hazards

TABLE 5—Demographic Characteristics of Food Pantry Clients According to Food Stamp Use

	Upstate				New York City			
	+ FS (n = 176)	−FS (n = 135)	Test Statistic	p	+FS (n = 105)	−FS (n = 103)	Test Statistic	p
			% of Responses					
Female respondent	63	37	8.925[a]	0.003	55	45	2.806[a]	0.094
Age of respondent (years)								
35 or under	61	39			53	47		
36–59	61	39			51	49		
60 or older	26	74	17.067[b]	0.0001	44	56	1.107[b]	0.575
Living/employment situation								
Live alone	61	39	0.779[a]	0.377	44	56	1.934[a]	0.164
With children	64	36	12.279[a]	0.0001	59	41	3.144[a]	0.076
Someone employed	38	62	17.989[a]	0.0001	24	76	15.572[a]	0.0001
Below 50% of poverty	46	34	4.053[a]	0.044	50	37	3.250[a]	0.071
Below 100% of poverty	93	70	26.552[a]	0.0001	90	71	10.651[a]	0.001
Median % of poverty	51%	74%	3.629[c]	0.0003	50%	68%	1.947[c]	0.051
Average monthly food stamp Benefit:								
(Household)	\$129 ± 80[d] (\$107–149)[f]	—	—	—	\$95 ± 76[d] (\$69–121)[f]	—	−3.467[e]	0.0006
(Per capita)[c]	\$43 ± 4[d] (\$39–47)[f]	—	—	—	\$37 ± 5[d] (\$32–42)[f]	—	2.155[e]	0.032

[a] χ^2 (df = 1).
[b] χ^2 (df = 2).
[c] Wilcoxon Rank Sum Test.
[d] Standard Deviation.
[e] t-value comparing Upstate vs NYC.
[f] 95% Confidence Interval.

associated with poor nutrition. In concert with similar data from non-random studies done in New York State between 1985 and 1987,[8–10] and additional analyses of this study,[11] a picture emerges of a sizable gap between household needs and available resources, a gap that food pantries have helped to bridge. If private food assistance diminishes (as has been predicted), government aid to the poor must be expanded or the risk of decreased food security and poor nutrition will increase. ☐

Acknowledgments

This survey was supported by a grant from the New York State Department of Health, Bureau of Nutrition and the New York State Legislature.

We would like to thank Dr. Janice Dodds, Deborah Spicer, and Thomas Melnick, from the New York State Department of Health, for their support and assistance. We are grateful to the study coordinator, Janet Hiemstra, for her invaluable work, and to all the interviewers from Syracuse and New York City. Dr. Susan Borker (Syracuse University) was our statistical consultant and Dr. Diane Murphy (Syracuse University) assisted with the interviewer training. We thank the directors and staff at all participating food pantries throughout the state, and the individuals who agreed to answer our questions.

References

1. New York State Department of Health: 1988 Census of Emergency Food Relief in New York State. Albany, NY: Bureau of Nutrition, Nutrition Surveillance Program, 1989.
2. Bowering J, Clancy KL, Poppendieck J: Characteristics of a random sample of emergency food program users in New York. II. Soup kitchens. Am J Public Health 1991; 81:914–917.
3. Levine D, Ingram L (eds): Income and Poverty Statistics: Problems of Concept and Measurement. Report of a Workshop, Washington, DC: National Academy Press, 1988.
4. Shapiro I, Greenstein R: Holes in the Safety Nets: New York State. Washington, DC: Center on Budget and Policy Priorities, 1988.
5. New York State Office for the Aging. Sources of Income of Older New Yorkers, Albany, NY, 1987.
6. Shapiro I, Greenstein R: Making work pay: A new agenda for poverty policies. Washington, DC: Center on Budget and Policy Priorities, 1989.
7. Greenstein R: Myths and realities about American poverty. Food Monitor No. 43, p. 16, Winter 1988.
8. Eldred CA, Satin MS: Characteristics and use of conventional benefits by users of soup kitchens and food pantries. City of New York Human Resources Administration, 1986.
9. Kelly G, Rauschenbach B, Campbell C: Private Food Assistance in New York State: Challenges for the 1990s. Ithaca, NY: Division of Nutritional Sciences, Cornell University, 1989.
10. Hunger Action Network of New York State. Food with Dignity: A Study of People Using Food Pantries in New York State. Albany, NY, 1987.
11. Clancy K, Bowering J, Poppendieck J: Audit and evaluation of food program use in New York state. Report on phase one. Report presented to the New York State Legislature and the New York State Department of Health, March 1989.

Characteristics of a Random Sample of Emergency Food Program Users in New York: II. Soup Kitchens

ABSTRACT

A random sample of soup kitchen clients in New York City was studied and specific comparisons made on various parameters including homelessness. Compared with the general population of low income persons, soup kitchen users were overwhelmingly male, disproportionately African-American, and more likely to live alone. The homeless (41 percent of the sample) were less likely to receive food stamps or free food, or to use food pantries. Fewer of them received Medicaid or had health insurance. Forty-seven percent had no income in contrast to 29 percent of the total sample. (*Am J Public Health*. 1991;81:914–917)

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Introduction

Soup kitchens, charitable organizations that provide free meals for consumption on the premises, were once regarded as relics of the depression era, useful only to serve the victims of disasters or the disreputable poor in the skid row sections of large cities. Since the early 1980s, however, soup kitchens have again become a significant source of food for the poor.[1]

In New York State, where a recent census identified 189 soup kitchens in New York City (NYC) and 138 in upstate New York and Long Island (Upstate),[2] the State Department of Health provides some funding for soup kitchens through the Supplemental Nutrition Assistance Program[3]; thus the future of the kitchens has become a question for public policy. This report presents data from a survey of persons utilizing soup kitchens in New York City. Upstate kitchens were not surveyed because they had recently been studied.[4]

Methods

The survey of 263 soup kitchen clients from 21 sites in New York City was conducted simultaneously with the food pantry study.[5] A random sample of kitchens was drawn from the Bureau of Nutrition census.[2] Within each site, clients were identified through a random sam-

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TABLE 1—Characteristics of Soup Kitchen Clients

	% of Responses				
	Entire Sample (n=263)	Homeless (n=108)	Not Homeless (n=155)	χ^2 Value	p
Gender					
Female	23	13	30		
Male	77	87	70	10.767[a]	0.001
Age of Respondent (years)					
35 or under	57	64	52		
36–59	36	35	37		
60 or older	7	1	11	9.592[b]	0.008
Ethnicity					
African-American	76	69	82	6.216[a]	0.013
Hispanic	16	23	11	6.915[a]	0.009
White	5	6	3	1.520[a]	0.218
Other[c]	3	2	4	—	—
Living Situation					
Alone	62	95	39	85.084[a]	0.0001
With Children	13	0	22	25.250[a]	0.0001
Someone in Household Employed	25	18	29	4.609[a]	0.032
Receive Food Stamps	32	20	40	11.282[a]	0.001
Receive Medicaid	27	15	36	14.411[a]	0.0001
No Medical Insurance	51	79	33	51.432[a]	0.0001

[a] Homeless vs Not Homeless; Chi-square (df=1)
[b] Homeless vs Not Homeless; Chi-square (df=2)
[c] Includes Asians and Native Americans

TABLE 2—Income Sources of Soup Kitchen Clients

	% of Households[b]				
	Entire Sample	Homeless	Not Homeless	χ^2 Value[a]	p
No Income	29	47	16	26.124	0.0001
Wages	22	17	26	3.093	0.079
AFDC (Aid to Families with Dependent Children)	5	1	8	6.929	0.008
Other Public Assistance	9	6	11	2.432	0.119
SSI/Other Disability	8	2	13	10.142	0.001
Social Security	8	3	12	7.543	0.006
Home Relief	7	3	10	5.383	0.020
Other[c]	7	2	11	—	—

[a] Homeless vs Not Homeless; Chi-square (df=1)
[b] Percentages do not add up to 100% because more than one response was possible
[c] Includes Veteran's benefits, Child support/alimony, Retirement/pension, and Unemployment benefits

pling procedure while waiting in line, but the interviews took place either before or after eating with as much privacy as possible. Interviewer selection and training were similar to the food pantry study.[5] The numbers of meals served by each site ranged from a few to 209,000 annually. As with the pantries, large and small sites were appropriately represented in the sample.[5]

Results

Eighty percent of clients had used soup kitchens for less than a year; the remainder were long-term users. Table 1 summarizes other characteristics of soup kitchen users in our sample. Compared with the general population of not homeless persons living below the poverty line in New York City[6] the soup kitchen users we studied were overwhelmingly male, disproportionately African-American, not elderly, and far more likely to live alone. Among those who did live with others, soup kitchen users (both homeless and not homeless) were much less likely than members of the general poverty population to live in households with children.

Compared to the not homeless, the homeless (the 41 percent of our sample who lived in shelters, or in the streets, or "no one place") were more likely to be male, younger, Hispanic, living alone, and unemployed. Fully 47 percent of them had no income.

Twenty-two percent of the entire sample earned wages (Table 2) and there was no significant difference on this between homeless and not homeless. Twenty-nine percent of the sample received welfare benefits, and the homeless were significantly less likely to receive assistance, including SSI and Social Security. The median monthly per capita income (calculated without food stamps) of the entire sample was $120, with 61 percent of them below 50 percent of poverty. The

TABLE 3—Income Distribution and Relation to Poverty Level

	% of Responses				
	Entire Sample (n=243)	Homeless (n=103)	Not Homeless (n=140)	Test Statistic	p
Household Income[a]					
$0	29	47	16		
$1–300	31	38	25		
$301–600	24	9	34		
$601–1000	9	5	13		
$> 1000	7	1	12	50.135[b]	0.0001
Income < 50% of Poverty	61	80	48	25.227[c]	0.0001
Income < 100% of Poverty	83	91	78	7.754[c]	0.005
Median % of Poverty	32%	3%	56%	−6.184[d]	0.0001
Median Monthly per Capita Income					
Excluding Zero Income[e]	$200	$164	$250	−2.192[d]	0.028
Including Zero Income	$120	$ 12	$186	−5.242[d]	0.0001

[a] Income excludes food stamps
[b] Homeless vs Not Homeless; Chi-square (df=4)
[c] Homeless vs Not Homeless; Chi-square (df=1)
[d] Homeless vs. Not Homeless; Wilcoxon Rank Sum Test
[e] Excludes 71 persons with zero income

TABLE 4—Demographic Characteristics of Food Stamp Users (+ FS) and Non-Users (− FS)

	% of Responses							
	Homeless				Not Homeless			
	+ FS (n=22)	− FS (n=86)	χ^2 Value	p	+ FS (n=62)	− FS (n=93)	χ^2 Value	p
Gender								
Female	14	86			40	60		
Male	21	79	0.367[a]	0.545	40	60	0.005[a]	0.943
Age of Respondent (years)								
35 or under	19	81			31	69		
36–59	23	77			49	51		
60 or older	0	100	0.491[b]	0.782	50	50	5.200[b]	0.074
Living/Employment Situation								
Live Alone	19	81	1.245[a]	0.264	51	49	4.906[a]	0.027
With children	0	0	—	—	38	62	0.839[a]	0.360
Someone employed	32	68	1.717[a]	0.190	20	80	10.498[a]	0.001

[a] Food Stamp Users vs Non-Users; Chi-square (df=1)
[b] Food Stamp Users vs Non-Users; Chi-square (df=2)

median monthly income of homeless respondents was $12 (Table 3) and 80 percent had incomes below 50 percent of the poverty level.

Approximately 30 percent of respondents received food stamps. Significantly more of the not homeless participated in the program (Table 1) but, among the homeless, there were no significant demographic differences between food stamp users and non-users (Table 4). In the not-homeless population, significantly fewer recipients lived with others, or in households where someone was employed (Table 4). The average monthly per capita food stamp benefit was significantly higher for the homeless recipients ($73 vs $40, t=−5.087, p=0.0001), undoubtedly reflecting their lower incomes.

Discussion

Comparison of this study with the few recent non-random studies,[7,8] the 1987 study of upstate New York soup kitchen clients,[4] and our food pantry study[5] was done to corroborate the extent of the problems being faced by low income persons. Except for a greater percentage of White clients, data gathered by the Human Resources Administration in 1986 are virtually identical to ours with respect to percent of males, age distribution, homelessness, lack of income, and receipt of public assistance, specifically SS and SSI.[7]

The biggest difference between soup kitchen clients in our NYC sample compared to Upstate clients[4] was the percentage of homeless (41 percent in NYC and 17 percent Upstate). Homelessness in NYC was also reflected in other differences: less use of food stamps, housing subsidies, and health insurance. The NYC clientele was younger, and many fewer received Social Security. More clients Upstate (more of whom were female) were disabled, retired, and received welfare benefits (AFDC, Home Relief) or SSI.

When NYC soup kitchen clients were compared with NYC food pantry users studied at the same time,[5] there were expected differences in age, ethnicity, and homelessness. The educational level was higher among soup kitchen respondents. Significantly fewer soup kitchen clients utilized food stamps or Medicaid, but there were no significant differences in the use of child nutrition and senior feeding programs.

There was a highly significant difference in the percentage of soup kitchen clients who had no income (29 percent) compared to food pantry users (8 percent). Since 88 percent of homeless soup kitchen users received no public assistance, 80 percent received no food stamps, and 79 percent had no health insurance, the precariousness of their lives is evident. A recent report from the National Academy of Sciences documents the fact that homeless people suffer more illnesses and injuries and are at greater risk for a variety of health problems including malnutrition.[9] Soup kitchen clients often eat only once or twice per day and soup kitchen meals tend to be nutritionally inadequate.[10]

Most of the clients in soup kitchens are obviously eligible for public assistance but do not receive it. Many have been unable to meet paperwork requirements; recommendations have been made for reducing these barriers so that more people can receive benefits.[6] However, full public assistance payments will not cover housing costs for most clients. The New York State courts have recently ruled that the Commissioner of Social Services must establish housing allowances that bear a "reasonable relation" to housing costs.[11] For many soup kitchen clients, adequate housing subsidies would provide more funds for food. For others, who are alcohol or drug abusers, other programs are necessary. In the meantime, the need for meals is growing and the size of the problem is too large to be met by the uncoordinated, largely voluntary Emergency Feeding System. ☐

Acknowledgments

The survey was supported by a grant from the New York State Department of Health, Bureau of Nutrition and the New York State Legislature. See previous brief for further acknowledgments.

References

1. Cohen BE, Burt MR: Eliminating hunger: Food security policy for the 1990s. Washington, DC: Urban Institute, 1989.
2. New York State Department of Health: 1988 Census of Emergency Food Relief in New York State. Albany: NY, Bureau of Nutrition, Nutrition Surveillance Program, 1989.
3. New York State Department of Health: New York State Emergency Food Relief Programs and The Supplemental Nutrition Assistance Program (SNAP). Albany, NY: Bureau of Nutrition, 1988.
4. Rauschenbach BS, Frongillo EA, Thompson FE, Andersen EJY, Spicer DA: Dependency on soup kitchens in urban areas of New York State. Am J Public Health 1990; 80:57–60.
5. Clancy KL, Bowering J, Poppendieck, J: Characteristics of a random sample of emergency food program users in New York. I. Food pantries. Am J Public Health 1991; 81:918–921.
6. Rosenberg TJ: Poverty in New York City, 1985–88: The crisis continues. New York City: Community Service Society of New York Poverty Perspectives, 1989.
7. Eldred CA, Satin MS: Characteristics and use of conventional benefits by users of soup kitchens and food pantries. New York City: City of New York Human Resources Administration, 1986.
8. Lasdon DH, Tobis D: Below the safety net: A study of soup kitchen users in New York City. New York: Legal Action Center for the Homeless and New York University, 1987.
9. National Academy of Sciences, Committee on Health Care for Homeless People: Homelessness, Health, and Human Needs. Washington, DC: National Academy Press, 1988.
10. Morris PM: A report of the quality of meals served in soup kitchens in New York State. Ithaca, NY: Nutritionists Organized Against Hunger, 1988.
11. *Barbara Jiggetts et al, v. William Grinker,* State of New York Court of Appeals, Opinion, April 3, 1990.

Farm Tractors and Mandatory Roll-Over Protection Retrofits: Potential Costs of the Policy in New York

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ABSTRACT

Tractor roll-overs are the leading cause of fatal farm accidents, accounting for more than one-fourth of all agriculturally related deaths. Most of these deaths could be prevented if the tractors were equipped with roll-over protective structures (ROPS). This study estimates the number of tractors in New York without ROPS, projects their retirement, and then estimates the number of lives which would be saved if ROPS were retrofitted on old tractors. The basic costs associated with mandating ROPS are calculated from these estimates. The minimum economic cost of mandating ROPS is $511,136 per life saved for the retrofits, and an additional $253,254 per life saved for every $1 million spent annually on enforcement. It is concluded that a policy mandating ROPS on all tractors would be expensive, but should be considered with particular attention to the need for and cost effectiveness of enforcement. (*Am J Public Health*. 1991;81:921–923)

Introduction

Agriculture consistently has one of the highest fatal accident rates of any occupation,[1] and tractor roll-overs play a prominent role in such accidents. Tractors are associated with about 50 percent of fatal agricultural accidents,[2] with roll-overs typically accounting for over half of these.[3–6] Evidence from Sweden suggests that most of these roll-over fatalities could have been avoided if the tractors involved had been equipped with roll-over protective structures (ROPS) to prevent operators from being crushed by an overturn.[7]

Most farm tractors in the United States lack ROPS, with only 19 percent of tractors in Pennsylvania[6] and 15 percent in one New York county* so equipped. ROPS became available in the 1960s, but did not become standard equipment on many tractor models until the late 1970s and early 1980s. In 1985, manufacturers voluntarily agreed to make them standard equipment on all new tractors.

Various proposals have been made to make ROPS mandatory in the US.[4,8] This study analyzes the National Coalition for Agricultural Safety and Health (NCASH) proposal to offer farmers retrofit incentives for five years and require ROPS on all tractors after 10 years. The potential economic cost per life saved from such a policy in New York is estimated using cost-effectiveness analysis, a common approach to appraising public health policies.[9–15] The cost shifting effects of incentives are not addressed in this analysis because of the study's economic (as opposed to financial) focus.

Methods

There are few available data on the age distribution of tractors currently on US farms or their ROPS status. The magnitude of the effect of ROPS in reducing the number and severity of non-fatal injuries is uncertain, especially when the operator is not wearing a seat belt, although anecdotal evidence suggests almost all deaths are prevented.

For this study the potential number of tractors which would be retrofitted in New York State was estimated by interpolating from US Census of Agriculture, Farm and Industrial Equipment Institute, and Pennsylvania tractor data, using the 4 percent retirement rate implied by data in the Census of Agriculture. There are no available data about the percentage of tractors of different ages in New York without ROPS, so the results of a survey in Pennsylvania by Huizinga and Murphy[6] had to be used. The geography, agricultural production, and economic situation of farmers in Pennsylvania and New York are roughly similar, making their data a relatively good approximation for tractors in New York. A total of 59,873 tractors would need to be retrofitted according to these calculations. It was assumed that all tractors covered by the 10-year requirement would be retrofitted during the first five years of the program, when the incentives are in effect (see Figure 1).

An average of nine people a year die from tractor roll-overs in New York State.** The number of deaths which would be averted by the retrofit policy was estimated by combining this with the current percentage of tractors without ROPS and the estimated pace of retrofits, under

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*Unpublished study of Otsego County, New York. Cooperstown, New York: New York Center for Agricultural Medicine and Health.

From the New York Center for Agricultural Medicine and Health. Address reprint requests to Timothy W. Kelsey, PhD, Agricultural Economist, New York Center for Agricultural Medicine and Health, One Atwell Road, Cooperstown, NY 13326. Mr Jenkins is a statistician with the Center. This paper, submitted to the Journal April 17, 1990, was revised and accepted for publication January 23, 1991.

**Unpublished data on New York agriculturally related accidents. Cooperstown, NY: New York Center for Agricultural Medicine and Health.

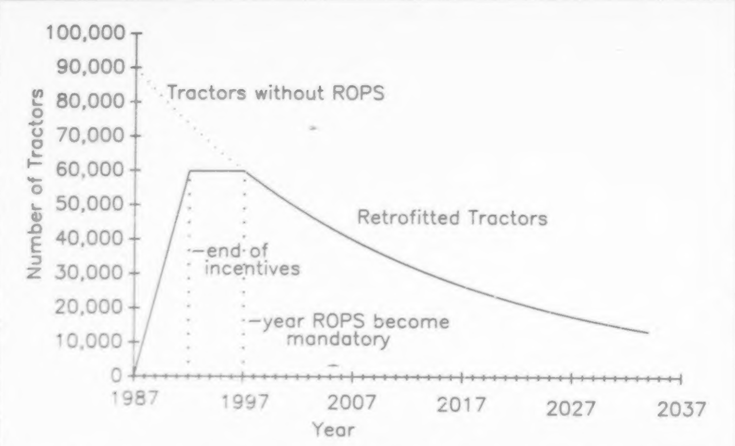

FIGURE 1—Estimated Number of Tractors Originally Manufactured Without ROPS Operating in New York if ROPS Became Mandatory on all New Tractors

TABLE 1—Cost Per Life Saved

Life-Saving Effectiveness of ROPS	Cost Per Life Saved* For ROPS	For Every $1 Million Spent Annually on Enforcement
75 Percent	$684,729	$337,672
100 Percent	$511,136	$253,254

*Discount rate = 5 percent

two alternative assumptions about the life-saving effectiveness of ROPS; 100 percent and 75 percent.

The costs of such a policy would include enforcement, as well as purchase and installation of the ROPS. Without enforcement there is no guarantee that all tractors will be retrofitted or that retrofitted ROPS will not later be removed. In Pennsylvania, 38 percent of the tractors manufactured since 1986 do not have ROPS, even though beginning that year tractors without ROPS were sold only under special circumstances. This occurred in part because farmers removed the ROPS after purchasing the tractor.[6] The reasons for removals have not been comprehensively studied, although the inability to operate a ROPS equipped tractor in buildings with low ceilings is a common explanation. The costs of enforcement can be significant, but because the NCASH proposal lacks an enforcement plan such costs will not be directly integrated into the analysis.

The specific cost of retrofitting tractors was determined by calling five ROPS manufacturers and wholesalers, including John Deere, Inc, and Ford/New Holland. The contacts indicated that increased sales would not affect most prices. The average uninstalled retail price reported for most ROPS was $632, which means the average final per unit cost will be approximately $700 with installation. The present value of all the retrofits was calculated assuming that the retrofits occur evenly throughout the five-year incentive period. Both costs and the lives saved were discounted with a 5 percent discount rate.

Results

A policy mandating ROPS in 10 years requiring any tractor in New York to have ROPS to be operated and providing economic incentives for retrofits during the first five years of the program could have a total ROPS cost of approximately $36.3 million. The present value of lives saved would be 53 or 71, depending upon the life-saving effectiveness of ROPS. The costs per life saved under the different scenarios are in Table 1. If perfect compliance does not occur and fewer lives are saved, the enforcement cost per life saved would be higher.

Discussion

These estimates of cost do not include consideration of the effectiveness and cost of enforcement. In addition, the model of tractor retirements biases the number of lives saved upwards. Another upward bias in estimation of the lives saved is that we could not include the natural reduction in farmers' exposure to risk over the years as tractors are slowly shifted from field work to more stationary roles and their hours of use decrease. Finally, non-pecuniary aspects of requiring ROPS retrofits are not included in these estimates.

Based on the projected cost without enforcement, ROPS would be similar to other life-saving interventions, but the need for enforcement could drastically increase cost. One reviewer found costs less than $1 million for auto safety standards and greater than or equal to $3 million for most efforts to regulate toxic substances.[16] Graham and Vaupel reviewed 35 studies of life-saving programs, and found the cost per life saved ranged from no cost for compulsory seat belt use, $60,000 for mandatory smoke detectors, $255,000 for the 1966 Motor Vehicle Safety Act, $408,000 for mandatory air bags, and $1,200,000 for the 55 mph speed limit, to over $3,500,000 for policies regarding toxic substances.[17]

Mandating ROPS retrofits is worth considering because of its life-saving impact, but enforcement must be directly and carefully debated because the policy impact will depend so much upon it and because enforcement could easily be the largest expense. In any event, a retrofit policy will not be cheap. ☐

Acknowledgments

The authors wish to thank David S. Pratt, MD, David Schweikhardt, PhD, John J. May, MD, Laura Marvel, RN, Judy Dennis, and Janet Ivory for their assistance in this project.

References

1. National Safety Council: Accident Facts. Chicago, IL: NSC, Various Years.
2. Purschwitz MA, Field WE: Scope and magnitude of injuries in the agricultural workplace. St. Joseph, MI: American Society of Agricultural Engineers, Paper No. 87-5514, 1987.
3. Piercy LR, Stallones L: Fatal Accidents on Kentucky Farms. St. Joseph, MI: Ameri-

can Society of Agricultural Engineers, Paper No. 84-5508, 1984.

4. Karlson T, Noren J: Farm tractor fatalities: The failure of voluntary safety standards. Am J Public Health 1979; 69:146–149.
5. Goodman RA, Smith JD, Sikes RK, Rogers DL, Mickey JL: Fatalities associated with farm tractor injuries: An epidemiological study. Public Health Rep 1985; 100:329–333.
6. Huizinga MA, Murphy DJ: Farm Work Injuries in Pennsylvania. Extension Circular 370. University Park, PA: College of Agriculture, Cooperative Extension Service, Pennsylvania State University, 1989.
7. Thelin A: EPILOGUE, Agricultural Occupational and Environmental Health: Policy Strategies of the Future. Am J Ind Med 1990; 18:523–526.
8. The National Coalition for Agricultural Safety and Health: Agriculture at Risk: A Report to the Nation. Iowa City: Institute of Agricultural Medicine and Occupational Health, University of Iowa, 1989.
9. Graham JD, Vaupel JW: Value of a life: What difference does it make? Risk Anal 1981;1:93.
10. Warner KE: Issues in cost effectiveness in health care. J Public Health Dent 1989; 49(Special Issue):272–278.
11. Javitt JC, Canner JK, Sommer A: Cost effectiveness of current approaches to the control of retinopathy in Type I Diabetics. Ophthalmology 1989; 96:255–264.
12. Goldman L: Cost-effectiveness perspectives in coronary heart disease. Am Heart J 1990; 119:733–740.
13. Simel DL, Matchar DB, Piscitelli JT: Routine intravenous pyelograms before hysterectomy in cases of benign disease: Possibly effective, definitely expensive. Am J Obstet Gynecol 1988; 159:1049–1053.
14. Martens LL, Rutten FFH, Erkelens DW, Ascoop CAPL: Clinical benefits and cost-effectiveness of lowering serum cholesterol levels: The case of simvastin and cholestyramine in the Netherlands. Am J Cardiol 1990; 65:27F–32F.
15. Cummings SR, Rubin SM, Oster G: The cost-effectiveness of counseling smokers to quit. JAMA 1989; 261:75–79.
16. Mendeloff JM: The Dilemma of Toxic Substance Regulation. Cambridge, MA: MIT Press, 1988.
17. Graham, Vaupel: *op. cit.* pp. 89–95.

The Use of Direct Mail to Increase Clinician Knowledge: An Intervention Study

Donald Sadowsky, DDS, MPH, PhD, and Carol Kunzel, PhD

ABSTRACT

A probability sample of American general practitioner dentists, 40 years of age or older, in solo private practice, was the target of two direct mail interventions offered at two different times, to test whether knowledge regarding prophylaxis of patients at risk for infective endocarditis could be improved. Tests of knowledge were responses in a mail questionnaire to clinical vignettes, designed to elicit the content of antibiotic regimens used for patients at risk. The research design enabled detection of (1) the effect of the interventions; (2) the differences in their effect; (3) the attenuation of their effect; and (4) the effect of time. Where baseline knowledge was low, it was improved and did not rapidly disappear. Both mail interventions were equally effective, in most instances, and there was no attenuation of the interventions' effect and no effect of time on the control groups' knowledge over a 4½ month period. The results suggest that it is possible to improve clinicians' knowledge of expert recommendations through direct mail intervention. (*Am J Public Health.* 1991;81:923–925)

Introduction

Direct mail has been used relatively infrequently, except by the pharmaceutical industry, to "market" ideas to clinicians. The results of several studies[1-7] testing the efficacy of mailed information in improving clinicians' knowledge are equivocal. Moreover, the effects of these efforts are often difficult or impossible to discern because of the absence of a sound experimental design.

Since the mid-1950s, the American Heart Association (AHA) has been trying to inform physicians and dentists about the details of a variety of antibiotic regimens that have been determined to be efficacious in the prevention of infective endocarditis, depending on the risk factor and the procedure to be performed. As the state of the art has changed, these AHA recommendations have been revised.[8-13] Dentists have been a target group for this AHA program because any procedure that produces mucosal bleeding may produce a transient bacteremia which, in turn, may seed a heart valve previously scarred by, for example, rheumatic heart disease. The ultimate sequela of this series of events may be infective endocarditis.

Our project had four aims: (1) to determine whether simple inexpensive mail interventions could be designed to improve the knowledge of dental general practitioners (GPs) about the management of patients at risk for infective endocarditis; (2) to design two interventions, one with a theoretical component and one without it, to determine whether the additional material would have an additional effect; (3) to determine whether the effects of the interventions, if any, would be maintained over time; and (4) to determine whether clinicians' knowledge would change over time, independent of the planned interventions.

Methods

After a probability sample of 1148 dentists, classified as GPs, 40 years or older, in private practice within the continental United States, was drawn from

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This paper was submitted to the journal January 29, 1991, and accepted with revisions March 7, 1991.

TIME	C1	C2	E1	E2	E3	E4
TIME+ ZERO T_0	No Stimulus	No Stimulus	Receives Recs (Intervention 1)	Receives Recs (Intervention 1)	Receives Recs + Theory (Intervention 2)	Receives Recs + Theory (Intervention 2)
No measurement for any group at this time						
TIME ONE T_1	Receives DCI*		Receives DCI* $\overline{E1} - \overline{C1}$ Equals Effect of Recs		Receives DCI* $\overline{E3} - \overline{C1}$ Equals Effect of Recs + Theory	
(4 1/2 Mos. Later)					$\overline{E3} - \overline{E1}$ Equals Effect of Theory	
TIME TWO T_2		Receives DCI* $\overline{C2}_{T2} - \overline{C1}_{T1}$		Receives DCI* $\overline{E1}_{T1} - \overline{E2}_{T2}$		Receives DCI* $\overline{E3}_{T1} - \overline{E4}_{T2}$
(Nine Mos. Later)		Test for "Natural History" Effect		Test for Attenuation of Recs		Test for Attenuation of Recs + Theory

*Data Collection Instrument

$\overline{C1}, \overline{C2}, \overline{E1}, \overline{E2}, \overline{E3}, \overline{E4}$ = Mean Knowledge Level of Group

+ Knowledge Level of All Six Groups Assumed Equal at Time Zero

FIGURE 1—Groups (Each group is representative of solo US GPs, 40 years or older and in private practice)

the American Dental Association's master list containing names of all dentists in the United States, the list was cleaned and criteria for eligibility were ascertained via postcards. Remaining potential respondents (686 GPs, 40 years of age and older, in solo private practice*) were randomly assigned to one of six experimental conditions. An overall response rate of 87.20% was achieved.

Four groups were contacted at baseline time zero (Fig 1). Two of these groups received a laminated placard (intervention 1), containing AHA information.[17] The reverse side of the placard was blank. The other two groups received the same placard but with 11 statements on the other side providing information about the theoretical underpinning of the AHA recommendations (intervention 2) with the expectation that enhancing understanding might help increase knowledge.[16] All placards were mailed with a cover letter encouraging the ongoing use of the placard as a resource in the office setting. Knowledge is used in this article to indicate availability of information. The remaining two groups (controls) were not contacted at time zero.

At time 1, 4½ months later, members of one control group, one group who had received intervention 1, and one group who had received intervention 2 were mailed an 8-page data collection instrument, along with a cover letter and a check for 10 dollars. At time 2, 4½ months after time 1, the same procedures were repeated for a second control group, for another group who had received intervention 1, and for a second group who had received intervention 2.

A precoded, forced choice, data collection instrument used varieties of clinical vignettes to elicit information. Dentists were asked to indicate the content of the antibiotic prophylactic regimen they might prescribe or administer for patients at different types of risk. Answers to questions about different patient conditions were assessed as "correct" = 1; "incorrect, unsure" = 0; "blank" = missing, using the standard of the most recent (at the time of the study) AHA recommendations. The resultant 0–1 dichotomous variables were analyzed as a special case of proportions, using either three-group or two-group χ^2 tests. The Yates correction was used for all two-group comparisons. SPSS/PC was used to perform all statistical computations.

Results

There was no significant attenuation in knowledge from one time period to the next in groups receiving the interventions, and knowledge scores remained stable over time in the control groups.

Because analyses indicated that time itself had no effect on knowledge scores, data for both control groups, for both intervention 1 groups, and for both intervention 2 groups were combined to ascertain the net effect of the interventions. χ^2 analyses contrasted the proportion of correct answers in the three types of experimental groups. Results indicate that, with the exception of the valvular heart disease patient, experimental group membership had a statistically significant impact on the proportion of correct answers about regimen components, as well as a statistically significant impact on correct answers about bacteremia duration, an important theoretical variable underlying the recommendations (Table 1).

There were no statistically significant differences between the two interventions for knowledge about the prosthetic heart disease patient, the child valvular heart disease patient, and the prosthetic heart valve patient with a penicillin allergy. Intervention 1 did not affect correct knowledge of managing the patient on a continuing antibiotic regimen and of bacteremia duration. However, intervention 2 had a statistically significant impact on both these knowledge items.

Discussion

The relative absence of an effect from the addition of scientific justifications for the AHA recommendations (intervention 2) can perhaps be explained by the findings of a comprehensive study by the RAND Corporation,[18] i.e., that consensus statements "are more likely to be effective if they are succinct, didactic, and oriented toward a clinical rather than a scholarly audience." The RAND study also provides a likely explanation for the uneven effects of the interventions: the negligible effect of the interventions on knowledge about the management of patients with a history of valvular heart disease is probably accounted for by the relatively high knowledge about antibiotic management of patients with this particular risk factor before the interventions. In reverse, this may also be a partial explanation for why knowledge about both the management of children and about patients with prosthetic heart valves improved considerably: for these conditions, baseline knowledge was very low and there was an opportunity for a great deal of improvement.

The improved knowledge scores of groups receiving intervention 2, on items about bacteremia duration and the man-

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*Eighty percent of American dentists are GPs[14] and, on a test of knowledge of endocarditis prevention, they scored dramatically lower than a cohort of dental specialists.[15] Also, dentists in solo general practice, 40 years or older, are most likely to be deficient in knowledge about the proper management of patients at risk for infective endocarditis.[16]

TABLE 1—Net Effect of Interventions (Time 1 and Time 2 Groups Combined)

Type of Patient and Knowledge Item Tested	Percentage of Correct Answers, %(n)				
	Controls	Intervention 1	Intervention 2	χ^2 df = 2	2-Tailed P[*]
VHD[a] patient					
Preprocedure drug	85 (149)	91 (169)	84 (156)	4.319	.115
Preprocedure dose	59 (110)	69 (135)	63 (118)	3.976	.137
When to begin Rx	68 (127)	72 (144)	73 (138)	1.502	.472
PHV[b] patient					
Preprocedure drug	15 (26)	28 (49)	23 (40)	8.329	.016
Preprocedure dose	14 (25)	25 (48)	26 (47)	9.339	.009
When to begin Rx	17 (32)	29 (58)	25 (46)	7.855	.020
80 lb VHD[a] Child					
Preprocedure dose	28 (53)	41 (82)	44 (85)	11.620	.003
Patient on continuing regimen of antibiotics					
Alteration in Antibiotic regimen required	32 (58)	32 (65)	43 (82)	7.225	.027
PHV[b] allergic patient					
Preprocedure drug	10 (19)	22 (43)	19 (36)	9.640	.008
Bacteremia duration	22 (41)	22 (44)	38 (74)	17.901	.000

[a] Valvular heart disease.
[b] Prosthetic Heart Valve.
[*] 2-tailed test was used because direction of change among three groups could not be anticipated.

agement of patients on a continuing antibiotic regimen, can be explained by the theoretical nature both of these items and of the intervention.

The experiment described provides evidence to support the notion that it is possible to improve clinicians' knowledge through direct mail intervention and that the effect does not rapidly disappear. More work must be done before the conditions under which this can be accomplished will be specified with any precision. Our study may provide some helpful hints about what works and what does not work. ☐

Acknowledgments

We wish to thank the National Institute of Dental Research for its support in this research (Grant No. DEO5622) and Dr. Rosalie Landres for her editorial assistance.

References

1. Soumerai SB, McLaughlin TJ, Avorn J. Improving drug prescribing in primary care: a critical analysis of the experimental literature. Milbank Q. 1989; 67:268–317.
2. Soumerai SB, Avorn J. Principles of educational outreach ('academic detailing') to improve clinical decision making. JAMA. 1990; 263:549–556.
3. Nazarian LF, Maiman LA, Becker MH. Recruitment of a large community of pediatricians in a collaborative research project. Clin Pediatr Phila. 1989; 28:210–213.
4. Lieberman HM, Emmett WL, Coulson AH. Pediatric automotive restraints, pediatricians, and the academy. Pediatrics. 1976; 58:316–319.
5. Harvey KJ, Stewart R, Hemming M, Naismith N, Moulds RRF. Educational antibiotic advertising. Med J Aust. 1986; 145:28–32.
6. Evans CE, Haynes RB, Birkett NJ, Gilbert R, Taylor DW, Sackett DL, Johnston ME, Hewson SA. Does a mailed continuing education program improve physician performance?: results of a randomized trial in antihypertensive care. JAMA. 1986; 255:501–504.
7. Jacoby I, Clark SM. Direct mailing as a means of disseminating NIH consensus statements: a comparison with current techniques. JAMA. 1986; 255:1328–1330.
8. American Heart Association: committee report on prevention of bacterial endocarditis. JAMA. 1990; 264:2919–2922.
9. American Heart Association: committee report on prevention of bacterial endocarditis. Circulation. 1984; 70:1123A–1127A.
10. American Heart Association: committee report on prevention of bacterial endocarditis. Circulation. 1977; 56:139A–143A.
11. American Heart Association: committee report on prevention of bacterial endocarditis. Circulation. 1972; 46:3–6.
12. American Heart Association: committee report on prevention of bacterial endocarditis. Circulation. 1965; 31:953–954.
13. American Heart Association: committee report on prevention of bacterial endocarditis. Circulation. 1955; 11:317-320.
14. American Dental Association. Distribution of Dentists by Region and State: 1987–1988. Chicago, Ill: American Dental Association; 1988, p 10.
15. Sadowsky D, Kunzel C. Clinician compliance and the prevention of bacterial endocarditis. J Am Dent Assoc. 1984; 109:425–428.
16. Sadowsky D, Kunzel C. Professional life cycle changes and their effect on knowledge level of dental practitioners. Soc Sci Med. 1989; 29:753–760.
17. American Heart Association Wallet Card, Pub. No. 78-004-D, 1-85-200M. (Available from the American Heart Association, Dallas, Tex)
18. Kanouse, DE, Brook RH, Winkler JD, et al. Changing Medical Practice Through Technology Assessment: An Evaluation of the NIH Consensus Development Program. The RAND Corporation, 1987, June. R-3452-NIH, 385,379.

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Postal Information: Second class postage paid at Washington, DC, and additional mailing offices. Postmaster: Send address changes to American Journal of Public Health, 1015 Fifteenth St., NW, Washington, DC 20005. AJPH allotted $40; TNH allotted $6 of APHA membership dues. Information on individual membership appears elsewhere in this Journal, or contact: Director, Membership Services, at APHA headquarters.

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Service Needs of Severely Disturbed Children

Eric W. Trupin, PhD, Amy Forsyth-Stephens, MSW, and Benson P. Low, PhD

ABSTRACT

Background: As one piece of a statewide Children's Mental Health System Analysis conducted in Washington State, the specific service needs of severely emotionally disturbed children were investigated.

Methods: The primary caseworkers of 3398 children under the care of the state responded to a survey of the child's treatment history, social and clinical condition, and service needs. Service need data on the 2455 children in the sample who were classifiable as severely emotionally disturbed were examined using log linear analysis.

Results: School- and home-based services were frequently reported needs of school-aged children. Adolescent children were reported to be in need of services which prepare them for adulthood. Services endorsed for female children tended to focus on assistance to family members, therapy, and protection, whereas services endorsed for male children focused on home removal, substance abuse treatment, and preparation for employment.

Conclusions: Severely emotionally disturbed children are in need of a variety of nontraditional mental health services. Significant differences in the needs of male versus female children, and of differently aged children, are present that have serious implications for service system development. (*Am J Public Health.* 1991;81:975–980)

Introduction

The treatment needs of severely and chronically mentally ill individuals, of any age, vary considerably depending upon personal characteristics (e.g., degree of disability, nature of symptomatology, willingness to accept treatment), and situational or environmental characteristics (e.g., availability of natural supports, residential situation, mobility of the individual). Ideally, the range of available services would be so broad, and care givers so flexible, that all clients would receive an appropriate, individualized menu of services. Unfortunately, human services, especially mental health, are largely program-driven, with clients matched to programs, rather than services matched to clients. Given that this infrastructure of care provision may be difficult to overturn, it is imperative that the development and design of state and local programs be based upon a concrete knowledge of client needs.

Services for children* are especially subject to "blind" planning and implementation. There is a dearth of research in the area of children's mental health, most notable on the topic of severely mentally ill children.[1] Analogous to the study of chronic mental illness among adults, the area of severe emotional disturbance (SED) among children is a relatively recently established field of research. Attention was drawn to the issue in 1982, when the unsettling book *Unclaimed Children*[2] reported that two-thirds of all SED children do not receive the services they need.

The book went on to document the failure of federal and state governments to acknowledge and attempt to meet the mental health needs of children, resulting in a vast and deeply troubled population with no advocacy, options, or hope. Five years later, the Office of Technology Assessment of the United States Congress[3] underscored accusations put forth in *Unclaimed Children.*

In 1984, the National Institute of Mental Health initiated the Child and Adolescent Service System Program (CASSP) in an effort to address the national crisis in children's mental health.[4] The goal of CASSP was, and continues to be, the promotion of system development through the funding of 40 state-level planning initiatives and two national research centers for the study of SED among children.

Most of the research to date on service needs of SED children has been produced by state human service departments in response to legislative mandates or class action suits, or by CASSP-funded research institutes. Based upon work completed by many states, and by the CASSP research centers, an assumption has been promulgated that a complete range of services, on a continuum from most to least restrictive, must be developed to adequately meet the needs of severely emotionally disturbed children.[5] Most recently, the "continuum of care" idea has been replaced by the concept of a "system of care" for SED children,[6]

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*In this article, "Children" refers to anyone under the age of 19 years.

which has a somewhat broader connotation. With the system perspective, more than a comprehensive spectrum of appropriate services is necessary. Just as critical are the underlying facilitating mechanisms, structures, and administrative processes to ensure that services are delivered and received in a coordinated, child-focused manner.

There have been no empirical studies of the diverse service needs of severely emotionally disturbed male children versus female children. Similarly,there have been no reports of service need data by developmental groupings (i.e. young children, preteens, and adolescents). Only one report has documented the age and sex breakdowns of an SED child population. In North Carolina, Behar[7] reports that the population of children qualifying as SED was 80 percent male, 20 percent female. Over three-fourths (78 percent) of the SED children were between the ages of 12 and 17; 14 percent were under the age of 12.

This paper presents the findings of a statewide survey of the specific service needs of 1617 severely emotionally disturbed children. The large sample size of the survey permits analysis of service need data by age and sex categories. The possibility of identifying distinct ideal service continuums for male and female SED children of various age groups is discussed.

Method

Design

The survey of service needs of SED children took place in the context of a broader study of the children's mental health system in Washington State. This larger project, entitled the Children's Mental Health System Analysis, involved the administration of a Child Mental Health Survey to adult care takers of randomly selected, nonidentified children served by the Department of Social and Health Services (DSHS).

The analysis of data occurred in two stages. The first stage involved the identification of children in the sample who were classifiable as SED (this procedure is described below under "Classification of Children as SED"). The second stage involved the examination of reported service need data for the subjects who were classified as SED according to our model.

A significant design feature was the nonidentified status of children surveyed. Use of identified data would have required informed consent for every child in the survey. Apart from the logistic demand such a requirement would have brought, institutional or agency refusals would have become a significant problem. While the use of nonidentified data facilitated more comprehensive surveying of children, it did limit control over the subject selection process, and precluded empirical analysis of subject attrition.

Subjects

The subjects were 3,398 children and adolescents ages 0 to 18 who were selected randomly from the billing rosters of child-serving programs within the State's Department of Social and Health Services (DSHS). Nearly half of the subjects ($N = 1,640$) were clients of the DSHS Division of Children and Family Services (DCFS), which offers an array of child welfare services including child protective services, foster care, group home care, in-home intervention, and emergency out-of-home placement. The remainder of the sample was made up of clients of the DSHS Division of Juvenile Rehabilitation (DJR; institutions, group homes, and parolees; $N = 1,035$); the DSHS Mental Health Division (DMH; inpatient, residential, day treatment, and outpatient; $N = 524$); and the DSHS Division of Developmental Disabilities (DDD; institutions and group homes; $N = 199$).

Sample sizes were calculated by taking into consideration the population size, the *a priori* estimate of the rate at which SED would occur (50 percent in all DSHS groups), the range of the intended confidence interval (+5 percent), and the probability level for the confidence interval (95 percent). Sample sizes were inflated by a factor of 15 percent, to compensate for subject attrition. As an exception, the DSHS Division of Juvenile Rehabilitation made an administrative decision to survey all clients in their programs. All clients in programs with caseloads of fewer than 150 were surveyed.

Respondents

The survey respondents were 3,398 adult DSHS professionals or staff members of agencies under contract with DSHS. Two-thirds of the respondents (65 percent) were case workers or facility staff employed either directly by DSHS, or by a contracted agency. The remaining one-third was made up of probation/parole officers and independent mental health professionals. Ninety-two percent of the respondents had at least a four-year college degree. The mean length of time that a respondent had known the surveyed child was 12.2 months.

Survey Instrument Development and Reliability

The data collection instrument, entitled the Child Mental Health Survey,* was developed after review of materials used by other states in similar but smaller-scale efforts.[8] The survey instrument contained six sections with questions pertaining to: 1) sociodemographic background; 2) exposure to 13 mental health risk conditions; 3) current behavioral adjustment as measured by the Child Behavior Checklist (CBCL);[9] 4) past mental health treatment and placement history; 5) other handicapping conditions; and 6) mental health and social service needs. The section on service needs required the respondent to identify the subject's past receipt, current receipt, and current unmet need for any of 32 human services.

The Child Mental Health Survey was administered to pairs of teachers of 26 special education pupils and 30 regular education pupils, and to pairs of case workers of 10 children placed in mental health treatment programs in the community. Each of the respondents independently completed surveys on the selected child. Reliability coefficients were calculated on four sections of the survey: exposure to mental health risk factors, mental health treatment history, other handicaps, and current behavioral adjustment. The Kappas obtained for the sections on risk factors, treatment history, and other handicaps were in the acceptable and expected range, with most values above .60, and some exceeding .80.[10]

Reliability coefficients were not calculated on service need data. Interrater reliability was less of a concern for the service need data, as the caseworkers who completed the survey forms were highly face valid, as they are the "natural" raters who drive the system of referrals for these SED children. Moreover, a meta-analysis on concordance of raters of children underscores that data from a single respondent typically provide a reasonable sample of what would be provided by other informants of the same type who see the child under generally similar conditions.[10]

Classification of Children as SED

SED children from the sample were identified via MANOVA techniques.

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*The Child Mental Health Survey is available from the first author upon request.

Each subject was treated as a group of one and then compared via Hotelling's T^2 against a criterion SED group. The criterion SED group was defined as all children being served in the State's psychiatric residential treatment centers, and a specialized foster care program for previously psychiatrically hospitalized children. These programs provide the most comprehensive, intensive, and expensive mental health treatment offered by the state mental health system for children. It was the research team's consensus that children meeting the numerous requirements for admission to these programs are inarguably severely emotionally disturbed, and therefore comprise the most valid, conservative, and defensible criterion group for this study.

Each subject was evaluated individually as to the likelihood of being drawn from the criterion group. If the subject was discriminable, the subject was not classified as SED. The independent variables used in the discriminant analysis were: 1) *Mental Health Risk,* scored as the number of risk factors endorsed positively for the subject; 2) *Chronicity,* scored as positive if the subject was rated as experiencing severe aggressiveness and/or withdrawal for a minimum duration of one year; 3) *Service Need,* scored as the number of mental heath services endorsed as "needed" or "currently received"; 4) *Internalizing Behavior Problem,* which was the T-score from the CBCL Internalization Scale; and 5) *Externalizing Behavior Problem,* which was the T-score from the CBCL Externalization Scale. These five measures are widely used and face valid with respect to the detection of the presence of an emotional or behavioral problem in children.[11–14]

Service Need Data Analysis

Service need data were analyzed for those children classified as SED according to the above model. Data on the services indicated as "needed" by SED children were weighted by DSHS population estimates of SED children across age and sex categories. This was done in order to control for the varying age and sex distributions of the four subsamples (i.e. DCFS, DJR, DDD, DMH). The population estimates were based upon the prevalence distribution of SED in our subsamples. A log linear analysis technique was used to test for significant differences in service need among three age groups of SED children (6–11, 12–16, and 17–18 years), and between male and female SED children.

Results

Survey Return Rates

The overall DSHS survey return rate was 75 percent (i.e. 4,516 surveys were distributed, 3,398 were completed and returned). Return rates for the various participating divisions of DSHS ranged from a high of 95 percent for DJR to a low of 66 percent for DCFS. The high overall survey return rate was primarily a function of consistent DSHS involvement with the design and execution of the study. Most supervisors at local DSHS offices required 100 percent survey completion by their caseworkers, and actively monitored this assignment. Additionally, researchers distributed two reminder notices to DSHS supervisors during the time frame allotted for survey completion. All respondents were assured of, and received, feedback on the outcome of the survey.

Service Needs of SED Children

Of our sample of 3,398 children, 2,455 were classified as SED according to our model (described above, $\alpha = .10$). Of these 2,455 SED children identified from our original sample, data on 1,617 were used in this analysis of service needs. A total of 838 cases were eliminated due to the subject's age (less than 6 years) and/or an incomplete survey form. Children ages 0 through 5 were not included in this analysis due to the small size of that subsample ($n = 96$). Table 1 reports the estimated percent of the DSHS SED population needing various mental health and social services.

Across all age and gender categories, over one-half of all SED children needed outpatient mental health treatment (defined as individual, group, or family therapy). Nearly one-half of SED children's families were in need of family support services for the parents and/or siblings. Interagency case management was needed by nearly one-third of SED children statewide. Other services which were commonly needed included school-based mental health services and psychological testing. Of all residential/inpatient services, foster home placement and group home placement were the most commonly reported service needs.

Age and Gender Service Need Differentials

Fourteen of the 32 possible mental health and social services listed on the Child Mental Health Survey showed significant differences in reported need across the three age groups. These are displayed in Table 2.

School children from age 6 through 11 (Group 1) were significantly higher than all other age groups in the need for respite care for their family members (11.0 percent, $p < .000$), and for therapeutic camp services (17.7 percent, $p < .000$).

Children in Age Groups 1 and 2 (ages 6–11 and 12–16) displayed a higher need for school and home-based services than older SED children, specifically school-based mental health services (Group 1: 30.0 percent; Group 2: 27.4 percent), supervised after-school activities (Group 1: 20.3 percent; Group 2: 18.0 percent), and intensive in-home therapy (Group 1: 15.6 percent; Group 2: 18.8 percent).

Children ages 12 through 16 (Group 2) were reported as more in need of foster home placements (18.9 percent, $p < .001$) and group home placements (12.6 percent, $p < .000$) than children in other age brackets. Data show that the need for both of these services peaks in the years 12 through 16, although the need pattern for the two services is quite different across the full developmental range. The need for foster home placement is high (12.3 percent) even within the 6 to 11 year old age group, and grows in the 12 to 16 year old group. At that point the need declines to 11.1 percent in the 17 to 18 year old age group. Group home placement, however, is low for the 6 to 11 year old age group, and then rises dramatically for the 12 to 16 year olds, staying relatively high (11.3 percent) for the 17 to 18 year old group.

Older adolescents (Group 3, ages 17–18) demonstrated a significantly higher need for outpatient alcohol or drug treatment (31.4 percent, $p < .000$), and inpatient alcohol or drug treatment (15.6 percent, $p < .000$) than the other age groups. They also demonstrated a much greater need than other age groups for training in independent living skills (58.6 percent, $p < .000$), vocational services (49.2 percent, $p < .000$), and supervised independent living arrangements (28.5 percent, $p < .000$). For each of these services, the expressed need was nonexistent or extremely low for younger children (ages 6–11), increased for children ages 12 through 16, and peaked for children 17–18 years of age.

Significant sex differences in endorsement of service need were identified among 11 of the 32 service types contained on the Child Mental Health Survey. These differences are also reported in Table 2.

Females were significantly higher than males in their reported need for out-

TABLE 1—Estimated Percent of Severely Emotionally Disturbed (SED) Child Population Needing Service, by Age and Gender

Service	Male Female Total	Ages 6–11 (N = 145) (N = 117) (N = 262)	Ages 12–16 (N = 594) (N = 284) (N = 878)	Ages 17–18 (N = 406) (N = 71) (N = 477)	All Ages (N = 1145) (N = 472) (N = 1617)
Outpatient Services					
Outpatient therapy	M:	60.5	49.3	32.1	49.6
	F:	72.1	63.0	44.9	63.2
	T:	66.0	55.9	37.3	56.0
Case management	M:	14.9	34.5	9.0	25.4
	F:	30.6	38.4	35.2	35.9
	T:	22.2	36.3	19.5	30.3
School-based mental health services	M:	33.9	27.5	9.9	26.4
	F:	25.5	27.4	22.3	26.3
	T:	30.0	27.4	14.9	26.3
Psychological testing	M:	29.1	27.3	22.8	27.1
	F:	15.7	24.4	15.9	21.1
	T:	22.9	25.9	20.0	24.3
Intensive in-home therapy	M:	15.2	17.0	9.8	15.4
	F:	16.1	20.9	15.9	19.0
	T:	15.6	18.8	12.2	17.1
Psychiatric evaluation	M:	15.8	20.4	16.7	18.6
	F:	14.3	14.5	10.6	14.0
	T:	15.1	17.6	14.3	16.5
Outpatient alcohol/drug treatment	M:	2.6	18.9	40.0	17.9
	F:	2.1	14.5	18.7	11.7
	T:	2.4	16.8	31.4	15.0
Day treatment	M:	11.9	9.1	7.1	9.5
	F:	4.9	6.1	7.7	6.0
	T:	8.6	7.7	7.3	7.9
Therapeutic preschool	M:	6.0	1.4	2.0	2.7
	F:	4.5	0.9	2.4	2.0
	T:	5.3	1.1	2.1	2.4
Medication evaluation	M:	3.4	3.3	0.7	2.9
	F:	2.3	1.7	5.4	2.3
	T:	2.9	2.5	2.6	2.6
Residential or Inpatient Services					
Foster home placement	M:	11.1	16.1	2.4	12.7
	F:	13.7	22.0	23.9	20.1
	T:	12.3	18.9	11.1	16.1
Group home placement	M:	3.5	15.3	13.3	11.9
	F:	3.8	9.7	8.3	6.0
	T:	3.6	12.6	11.3	10.1
Inpatient alcohol/drug treatment	M:	0.0	8.4	25.5	8.9
	F:	0.0	8.2	0.8	5.2
	T:	0.0	8.3	15.6	7.2
Crisis residential placement	M:	0.0	3.5	1.4	2.0
	F:	4.3	9.5	0.2	7.0
	T:	2.0	6.4	0.9	4.5
Psychiatric residential treatment	M:	2.2	2.7	2.8	2.6
	F:	1.0	2.9	5.1	2.7
	T:	1.6	2.8	3.8	2.6
Psychiatric hospitalization	M:	0.0	1.9	0.7	1.2
	F:	0.0	1.9	0.0	1.2
	T:	0.0	1.9	0.4	1.2
Services to the Family					
Family support group	M:	44.8	46.4	38.0	44.6
	F:	43.3	55.5	50.8	51.7
	T:	44.0	50.8	43.2	47.9
Respite care	M:	11.1	3.5	1.9	5.2
	F:	10.8	7.7	5.1	8.2
	T:	11.0	5.5	3.2	6.6
Services to Adolescents					
Training in independent living skills	M:	0.0	20.9	58.4	21.3
	F:	1.1	20.5	59.0	20.0
	T:	0.5	20.7	58.6	20.7
Vocational services	M:	0.0	18.5	56.3	19.5
	F:	1.1	16.2	38.6	14.9
	T:	0.5	17.4	49.2	17.4

continued

patient therapy (61.7 percent, $p < .000$), foster home placements (19.1 percent, $p < .001$), respite care (8.0 percent, $p < .000$), supervised independent living arrangements (7.6 percent, $p < .001$), and child protective services referral/intervention (9.6 percent, $p < .000$).

Males were rated as having a significantly greater need for outpatient alcohol or drug treatment (16.2 percent, $p < .000$), inpatient alcohol or drug treatment (7.9 percent, $p < .000$), group home placement (10.6 percent, $p < .000$), training in independent living skills (19.0 percent, $p < .000$), and supervised afterschool activities (19.8 percent, $p < .006$).

Discussion

Our survey of the professional caregivers of SED children generated a useful data base on the service needs of this population. While previous research and program planning efforts have identified only general categories of services appropriate for SED children, this broad-based study is the first to produce quantitative data on the extent of the SED population reported to be in need of a wide range of mental health, health, social, and support services. It is also the first study that further defines age and gender differences in types of needed services.

Overall Patterns of Service Need

Across age and sex groupings, the most frequently reported needs, in descending order, were for: 1) outpatient therapy; 2) family support groups; 3) intersystem case management; 4) school-based mental health services; 5) psychological testing; 6) training in independent living skills; 7) intensive in-home therapy; and 8) supervised after-school activities. With the exception of outpatient therapy and psychological testing, all of these services would be considered "non-traditional" mental health services, or even outside the realm of mental health altogether.[6]

We had anticipated that our survey of professionals would result in a high expressed need for residential or inpatient services, since respondents' level of familiarity with other more progressive treatment approaches was questionable. We reasoned that direct care providers might be hesitant to endorse non-traditional services as needed, especially if such services were not accessible to them.

Clearly, this was not the outcome. The strong presence of CASSP in Washington State may be credited for a good

TABLE 1—Continued

Service	Male Female Total	Ages 6–11 (N = 145) (N = 117) (N = 262)	Ages 12–16 (N = 594) (N = 284) (N = 878)	Ages 17–18 (N = 406) (N = 71) (N = 477)	All Ages (N = 1145) (N = 472) (N = 1617)
Supervised independent living arrangement	M:	1.0	5.1	27.1	7.5
	F:	0.0	7.9	30.5	8.5
	T:	0.6	6.4	28.5	7.9
Other Services					
Supervised afterschool activities	M:	22.4	22.8	10.7	20.8
	F:	17.9	12.8	2.3	12.9
	T:	20.3	18.0	7.4	17.1
Transportation to needed services	M:	22.7	13.9	5.1	14.8
	F:	12.4	15.0	5.5	13.1
	T:	17.9	14.4	5.3	14.0
Recreational services	M:	13.7	20.1	7.3	16.4
	F:	10.4	11.4	2.9	10.1
	T:	12.2	15.9	5.5	13.5
Therapeutic camp	M:	20.0	10.9	2.6	12.0
	F:	15.0	10.0	4.9	10.7
	T:	17.7	10.5	3.5	11.4
Special education	M:	15.3	12.4	12.7	13.2
	F:	6.3	10.3	3.0	8.4
	T:	11.1	11.4	8.8	11.0
Child protective services	M:	5.5	2.3	1.2	3.0
	F:	11.5	6.0	6.7	7.5
	T:	8.3	4.1	3.4	5.1
In-home education	M:	5.6	4.2	1.3	4.1
	F:	5.5	4.1	10.2	5.2
	T:	5.6	4.1	4.9	4.6

TABLE 2—Significant Age and Gender Effects

Service	Direction of Effect
Age Main Effects	
Therapeutic preschool	1 > 2,3
Respite care	1 > 2,3
Therapeutic camp	1 > 2 > 3
Transportation to needed services	1,2 > 3
Intensive in-home services	1,2 > 3
School-based mental health services	1,2 > 3
Foster home placement	1,2 > 3
Supervised after school activities	1,2 > 3
Group home placement	2,3 > 1
Outpatient alcohol/drug treatment	3 > 2 > 1
Inpatient alcohol/drug treatment	3 > 2 > 1
Training in independent living skills	3 > 2 > 1
Vocational services	3 > 2 > 1
Supervised independent living arrangement	3 > 2 > 1
Gender Main Effects	
Outpatient therapy	F > M
Foster home placement	F > M
Respite care	F > M
Supervised independent living arrangement	F > M
Child protective services	F > M
Outpatient alcohol/drug treatment	M > F
Inpatient alcohol/drug treatment	M > F
Group home placement	M > F
Training in independent living skills	M > F
Vocational services	M > F
Supervised after school activities	M > F

Note.
Group 1 = 6–11 year olds
Group 2 = 12–16 year olds
Group 3 = 17–18 year olds
All main effects $p < .01$

deal of the progressive thinking of the state's professional care givers through conferences, newsletters, and an intensive effort to begin the creation of local intersystem case coordination committees. Not recognizing the significant impact of these educational efforts, we underestimated the knowledge base of our respondent pool.

Unfortunately, Washington State's current service system reflects the national reality, in that treatment resources continue to be focused on a proportionally small number of children who receive care on inpatient psychiatric units or other intensive residential treatment programs.[3,15] Our data support the direction of CASSP-funded research and training centers and the National Mental Health Association in the endorsement of less restrictive home and school-based services, family support services, and comprehensive case management services as alternatives to costly out-of-home care.[15,16]

Age and Sex Differences

Service planners generally tend to think of SED children as one class of service recipients. Our data suggest that there are significant differences in the service needs of male versus female children, and of differently aged children. These differences have serious implications for the development and implementation of systems of care for the SED population.

Our data demonstrate a substantial developmental gradient which must be considered when planning services for SED children. For school-aged children the educational setting clearly should play a large role in treatment planning. For example, school-based mental health services and supervised after school activities were distinct needs of 6–16 years olds. Therapeutic camp services and foster home placements were also highly endorsed for this subset of SED children.

For the 17–18 year old group, there is a great need for services which prepare youth for the transition for adulthood, such as training in independent living skills, vocational services, and supervised independent living arrangements. The need for outpatient and inpatient alcohol/drug treatment also peaks for this age cohort.

Sex differences in service need were less obvious than the developmental differences. Males demonstrated a greater need for transitional "adult" services, for alcohol and drug-related treatment, and for group home placement. Females, on the other hand, were in greater need of outpatient therapy, foster home placements, respite care for their families, supervised living arrangements, and protective services. The services endorsed for males focused on home removal, treatment of addiction, and preparation for entry into the work force. Services endorsed for females focused on therapy, assistance to the girls' families, and protection from further harm. More research is needed to confirm whether these gender-linked service needs are valid indices of what SED children need in their treatment, or merely reflections of sex-based stereotypes of SED children's vulnerabilities and responsiveness to various treatments and interventions.

Study Limitations

Limitations of this study lie in its narrow focus on professional respondents, the non-random nature of the final sample, and the survey's exclusion of preventive services.

First, our survey respondents were exclusively the professional care givers of SED children. This clearly provides only one perspective on what a given child's treatment needs may be. Family members, school personnel, and other persons in the child's support circle are equally valid respondents. Nevertheless, given that the focus of the survey was on SED children who were "system veterans" with well-known and extensive patient histories, input from the primary professional care givers was a good starting point. This in no way precludes the need to vigorously solicit the participation of parents and teachers in the development of individual children's treatment plans and larger scale planning endeavors.

Second, for the analyses of service need data, the ultimate data base of SED children was a nonrandom approximate 50 percent of our original random sample of children. Cases were eliminated for three possible reasons: 1) the survey form was not returned; 2) the survey form was returned, but was missing critical information; or 3) the child was under 6 years of age. Ideally, our final sample would have been of a completely random nature. However, because the subjects were unidentified to the investigators, and since no special services were offered to children as a result of survey responses, the final sample appeared not to be biased systematically. Case worker-related factors, such as excessive paperwork demands, rather than child-related factors, appeared to contribute to the rate of subject loss.

Third, because children who were already classifiable as SED were the subjects of this study, primary preventive services were completely excluded from the range of services offered as possible service needs on our data collection instrument. Readers must keep in mind that the services highlighted as "most needed" by our survey represent the narrow but critical range of mental health and other services for those children who are already identified as SED. Services such as community education, parent training, teen parenting classes, substance abuse prevention, advocacy, early childhood intervention, etc., are very important elements of a comprehensive strategy to combat severe childhood mental illness. ☐

Acknowledgments

This research was supported in part by the Washington State Department of Social and Health Services, Mental Health Division, contract number 6500-62476, and National Institute on Disability and Rehabilitation Research grant number H133C80002.

References

1. Saxe L, Cross TP, Silverman N: Children's mental health: The gap between what we know and what we do. *Am Psychol.* 1988; 43:800–807.
2. Knitzer J: Unclaimed Children: The Failure of Public Responsibility to Children and Adolescents in Need of Mental Health Services. Washington, DC: Children's Defense Fund, 1982.
3. Saxe L, Cross TP, Silverman N: Children's Mental Health: Problems and Services. Durham, NC: Duke University Press, 1987. (Original work published by the Office of Technology Assessment, US Congress. Washington, DC: Govt Printing Office, 1986.)
4. National Institute of Mental Health: Program Announcement: Child and Adolescent Service System Program. Washington, DC: NIMH, December 1983.
5. Friedman RM: Major issues in mental health services for children. *Admin Ment Health.* 1986;14:6–13.
6. Stroul BA, Friedman RM: A System of Care for Severely Emotionally Disturbed Children and Youth. Washington, DC: CASSP Technical Assistance Center, Georgetown University, 1986.
7. Behar L: Changing patterns of state responsibility: A case study of North Carolina. *J Clin Child Psychol.* 1985;14:188–195.
8. Stroul BA: Child and Adolescent Service System Program State Needs Assessment Materials. Washington, DC: Georgetown University Child Development Center, CASSP Technical Assistance Center, 1985.
9. Achenbach TM, Edelbrock C: Manual for the Child Behavior Checklist and Revised Child Behavior Profile. Burlington, VT: Department of Psychiatry, University of Vermont, 1978.
10. Achenbach TM, McConaughy SH, Howell CT: Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psych Bull.* 1987;101:213–232.
11. Achenbach TM: Developmental Psychopathology. New York: Wiley, 1982.
12. Boyle MH, Offord DR, Hoffman HG, *et al:* Ontario child health study. *Arch Gen Psychiatry.* 1988;45:1120–1126.
13. Mooney KC, Thompson R, Nelson JM: Risk factors and the Child Behavior Checklist in a child mental health center setting. *J Abnorm Child Psychol.* 1987;15:67–73.
14. Garrison WT, Earls F: The Child Behavior Checklist as a screening instrument for young children. *J Am Acad Child Psychiatry.* 1985;24:76–80.
15. Zeigler-Dendy CA: Invisible Children Project: Final Report and Recommendations of the Invisible Children Project. Washington, DC: National Mental Health Association, 1989.
16. Research and Training Center for Children's Mental Health: Update: Improving services for emotionally disturbed children. Tampa, FL: The Center, Winter 1988–89.

Errata

In: Robinson JC, Paxman DG. OSHA's four inconsistent carcinogen policies. *Am J Public Health.* 1991;81:775–780. Public Health and the Law.

The names of the authors, J. C. Robinson and D. G. Paxman (and not the name of the column editor), should have been listed with the article's title on the table of contents. We regret the error.

* * *

In: Mandelblatt J, Andrews H, Kerner J, Zauber A, Burnett W. Determinants of late stage diagnosis of breast and cervical cancer: the impact of age, race, social class, and hospital type. *Am J Public Health.* 1991;81:646-649.

The word "non-public" should read "public" in three different places as follows: in Table 3, Profile 5; in Table 5, Profile 5; and in Table 5, Profile 6. In Table 5, footnote a), ".67" should read ".53." In footnote b) of the same table, ".35" should read ".13."

Analysis of US Child Care Safety Regulations

Carol W. Runyan, MPH, PhD, Diana E. Gray, MPH, Jonathan B. Kotch, MD, MPH, and Matthew W. Kreuter, MPH

ABSTRACT

Background: With 1.9 million US children cared for in organized group child care, the safety of these children is a public health concern. In the absence of federal policy, each state has developed its own day care safety regulations.

Methods: After creating a set of 36 criteria from three sets of national guidelines, we assessed the safety regulations of 45 states. With a mailed survey of state day care regulatory personnel, we examined the processes of formulating and implementing safety policy in 47 states.

Results: For 24 of the 36 items, more than half the states' regulations were below the criteria or failed to mention the topic. Most notable is the inattention to playground safety, choking hazards, and firearms.

Conclusion: The uneven quality of regulations may be a reflection of a regulatory process that is fragmented, with many different groups sharing authority and with limited involvement of injury prevention specialists. (*Am J Public Health*. 1991; 81:981–985)

Introduction

According to data from the winter of 1984–85, 1.9 million United States children of working mothers were cared for in organized group day care, and an additional three million received regular care in private homes other than their own.[1]

The importance of injuries to preschoolers is well-documented,[2–4] as are the patterns of some specific injury problems including poisoning,[5,6] aspiration,[7,8] falls,[2] drowning,[9] electrical shocks,[10] scalds and burns,[11] and injuries associated with playgrounds[12] and firearms.[13,14] Only a few studies have examined injury in US day care settings.[15–21] In the most comprehensive report to date, Sacks, *et al.*[15] found that 58 percent of all day care center injuries result from falls. Falls from playground equipment account for 48 percent of serious injuries, with the playground surface an important determinant of injury severity.

Despite the large numbers of children in organized child care, the incidence of childhood injury, and several legislative efforts, no federal day care regulations exist except for centers on military bases (70,000 children) and for Head Start programs (489,000 children).[16,17] Instead, each state formulates and enforces its own regulations. State day care regulations address many issues including disease transmission, hygiene, provision of nutritious and safe foods, provision of an environment which facilitates the child's mental, physical, and emotional growth, as well as injury prevention.

Our goal was to assess the content of state safety regulations and document how they are established and enforced.

Methods

The study consisted of three elements: 1) development of the set of model criteria to assess the regulations; 2) content analysis of the regulations; and 3) a questionnaire documenting day care regulatory processes in the states.

The reference criteria were drawn from three sets of nationally recognized guidelines: Massachusetts' Department of Public Health, *Safe Daycare: A Teacher's Guide for Creating Safe Environments for Preschool Children*[10]; American Academy of Pediatrics (AAP) *Health in Daycare: A Manual for Health Professionals*[24]; National Academy of Early Childhood Programs, *Accreditation Criteria and Procedures*.[23] From a combined list of 130 injury-related criteria, 36 specific items were selected by a panel of four injury specialists including three of the authors plus one independent reviewer. Selections were based on individual judgments of the panelists about the potential importance of the injury problem for young children but with an aim of including a diverse, if limited, set of categories. We specifically focused on regulations addressing environmental features of the facilities and center procedures rather than issues such as staff hiring and training practices or staff:child

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This paper, submitted to the Journal March 9, 1990, was revised and accepted for publication January 23, 1991.

ratios because the relationship between staffing patterns and injury potential is less well documented.

Content Analysis

The content analysis consisted of a comparison of each state's regulations to the 36 items. Each was rated as: 1) exceeding the criterion; 2) meeting the criterion; 3) being below the criterion; 4) not mentioning the content area; or 5) referring to other state regulations not specific to day care and not available to us (e.g. state building or fire codes). In the context of this study, we refer to day care as defined by the American Academy of Pediatrics[24] as "care, supervision, and guidance of a child or children, unaccompanied by parent or other legal custodian, on a regular basis, for periods of less than 24 hours." The content analysis, however, was restricted to analysis of regulations pertinent to day care center facilities. We excluded consideration of regulations specific to family day care as defined by the AAP; that is, "situations in which fewer than six children are cared for in the caregiver's home."[24] None of the criteria chosen delineated differences for specific age groups, although several of the criteria referred to issues specific to an age group (e.g. regulations about cribs).

Questionnaire

The questionnaire about the regulatory process and compliance was sent to the state agency responsible for regulating child day care at the same time the regulations were requested, by letter, from each state. The 15-item self-administered survey addressed the development and monitoring of safety regulations. Nonrespondents were phoned five weeks after the initial mailing and reminded of the survey.

Results

Analysis of Regulation Content

Regulations were obtained from 45 states and examined in comparison to the 36 criteria. Evaluations of the regulations, grouped by category, are presented in Table 1, demonstrating the proportion of states by whether each regulation exceeds, meets, or is below the criterion or if the topic is referred to in another code or not mentioned.

There were only five of the criteria (13.9 percent) that at least half the states met whereas 15 of the 36 items (41.7 percent) were not mentioned by half or more of the states.

For certain topics such as fire prevention, the state regulations frequently did not list specific requirements but referred to building codes or other state policies. Consequently, comparison to the criteria was impossible. For four of the six standards addressing fire and burn prevention, over half the states referred to other documents. At least 10 percent of the states referred to other regulations for swimming and wading pools, elevated areas, right hand descending stair railings, and motor vehicle passenger restraint.

Surprisingly, no state specified maximum heights for playground equipment, 96 percent neglected to mention playground surfacing, and 91 percent did not specify an eight-foot clearance between playground equipment.

There also were gaps in addressing indoor hazards. For example, 62 percent of the states were below the criterion in their restriction of objects that might cause choking, and no state specifically mentioned the most common causes of choking: hot dogs, peanuts, and grapes.[6,7] Only 26 percent of the states met the criterion for proper storage of poisonous substances, and a mere 11 percent specified that facilities not allow loaded firearms.

Although 55 percent of the states met or exceeded the criterion for tap water temperature, 38 percent failed to mention the issue. Eighty percent failed to meet the criterion that no free-standing space heaters be used.

Although 82 percent of states required that facilities have a first aid kit at the center at all times, only 18 percent required that at least one staff member be certified in cardiopulmonary resuscitation (CPR).

Subsequent to this analysis, the American Public Health Association (APHA) and the American Academy of Pediatrics (AAP) have drafted joint standards for child day care policy, including safety.[26] All of the 36 items included in our analysis are included in the AAP/APHA guidelines. For three of the 36 items, the AAP/APHA recommendations were more stringent than those used in this analysis, while for six others the AAP/APHA recommendations were slightly less stringent. For example, our analysis included a criterion that poisonous substances and medicines be kept in locked storage; the corresponding AAP/APHA guidelines indicated that there be "inaccessibility" of these substances.

The standards for Head Start programs were last revised in 1975 and indicate that if facilities meet or exceed state or local licensing requirements for fire, health, and safety this will "be accepted as prima facie compliance."[27] Our review of the 17 Head Start standards pertaining to safety indicated that none was more rigorous than the 36 criteria we considered.

Analysis of Perspectives on the Regulatory Process

Survey responses were received from 47 states (missing are Florida, Louisiana, and Maryland). Only four states *required* that injury prevention specialists be involved in developing regulations, although 16 states reported that injury prevention specialists are "routinely involved."

Safety regulation compliance by child day care centers was perceived to be good, with ratings of "better than average" reported by respondents from 26 states (55 percent). In contrast, compliance on the part of family day care homes was perceived to be "better than average" in 16 states (34 percent).

Respondents in 23 states indicated that at least one center had been closed in the prior year for safety violations and 19 reported that at least one family day care home had been closed. The types and number of inspections conducted by states varied widely. Types of inspections conducted included fire, gas pipe, sanitation, licensing, and routing monitoring; number of inspections per facility per year ranged from zero to six. Twenty-four respondents indicated that child day care centers in their states were inspected, on average, one or fewer times per year (e.g. every other year).

Those responsible for inspecting the facilities vary by state, with the jurisdiction for inspection shared by seven different types of groups or agencies. In some states, several different agencies were indicated as being responsible for development and/or enforcement of the regulations. In addition, respondents suggested that improvements were needed in: training for licensing and enforcement personnel; transportation policies; and attention to pets, electrical hazards, lead paint, and playground surfacing.

Discussion

Several limitations in the research should be noted. The content analysis was restricted to 36 specific day care center standards and to the 45 states providing

TABLE 1—Proportion of States by Level of Adherence to Criteria (n = 45)[a]

Criteria	Level of Adherence: Exceeds Criterion	Meets Criterion	Below Criterion	Topic not Mentioned	Refers to Other Code
Outdoor Hazards					
Wading pools emptied when not in use	2.2	24.4	11.1	44.5	17.8
All pools should be fenced & gates locked	6.7	20.0	17.8	24.4	31.1
Fences, natural barriers around play areas near busy streets, unsafe areas	0.0	73.3	6.7	20.0	0.0
Play equipment anchored, anchors below ground, no exposed concrete	0.0	6.6	35.6	57.8	0.0
At least 8 feet between all playground equipment	0.0	0.0	8.8	91.2	0.0
Maximum height of playground equipment 6 feet	0.0	0.0	0.0	100.0	0.0
8 inches or more of a resilient surface under playground equipment	0.0	0.0	33.3	66.7	0.0
Continuous maintenance of loose play surface materials	0.0	2.2	2.2	95.6	0.0
Flexible, light-weight, non-cutting swing seats	0.0	4.4	0.0	95.6	0.0
Structure of Facility					
Window openings limited to six inches	0.0	4.4	4.4	91.2	0.0
Elevated areas (landings, balconies, porches) provided with railings, adaptations, or approved safety gates	0.0	37.8	13.3	35.6	13.3
Right hand descending railing secured at child height	2.2	0.0	37.8	48.9	11.1
Above ground windows accessible to children must be constructed, adjusted, or adapted with window stops, screens, or grills	0.0	20.0	31.1	46.7	2.2
Indoor Hazards					
Non-metallic, tight-fitting caps in electric receptacles that are not in service in areas where young children are in care	0.0	55.5	0.0	44.5	0.0
Crib sides always up, in locked position when occupied	0.0	4.4	6.7	88.9	0.0
Toys, furnishings and equipment should be non-toxic, lead-free	0.0	48.9	33.3	17.8	0.0
Toys, furnishings and equipment should be free of rust, splinters and sharp corners	0.0	64.5	22.2	13.3	0.0
Toys, furnishings and equipment should be free from objects small enough to swallow	2.2	35.6	4.4	57.8	0.0
No peanuts, chunks of hot dogs, grapes	0.0	0.0	0.0	100.0	0.0
All potentially poisonous or hazardous substances kept in locked storage space, inaccessible to children	0.0	24.5	62.2	13.3	0.0
Rx/Non Rx medications kept in a designated locked place, inaccessible to children	0.0	28.9	60.0	8.9	2.2
No loaded gun permitted in child care facility	8.9	2.2	8.9	80.0	0.0
Fire/Burn Prevention					
Fire/Smoke Detector provided	2.2	37.8	2.2	0.0	57.8
Fire/Smoke Detector tested monthly (weekly)	2.2	6.7	2.2	17.8	71.1
Fire extinguishers provided	0.0	42.3	4.4	2.2	51.1
Fire extinguishers tested monthly (not specified)	0.0	4.4	15.6	15.6	64.4
Free standing space heaters should not be used	0.0	20.0	17.8	60.0	2.2
Hot water temperature should not exceed 120 degrees F at outlet	22.2	33.3	6.7	37.8	0.0
Emergency Procedures					
First Aid Kit in center at all times, wherever, whenever children are in care	0.0	82.2	8.9	8.9	0.0
At least one staff person on premises who is currently certified in CPR	2.2	15.6	8.9	73.3	0.0
Supervision					
Adult supervision of children at all times	0.0	91.1	8.9	0.0	0.0
No infant left unattended while bathing	0.0	11.1	0.0	88.9	0.0
Safety Education					
Safety education for parents	0.0	0.0	0.0	100.0	0.0
Safety education for children	0.0	4.4	2.2	93.4	0.0
Transportation					
All vehicle passengers seated in seats with suitable restraint system	0.0	22.2	31.1	26.7	20.0

[a] Data from the following jurisdictions were either not received or were not usable: Florida, Kansas, Louisiana, Maryland, Nevada and Washington, D.C.

documents. A separate analysis is needed to address the standards for the large population in family day care homes. The survey also was limited in several ways: only one representative responded from each state, and the role of these respondents may differ among states; anonymity was impossible, potentially reducing candor; and judgments of compliance were based on respondents' recall of "usual" practices which may vary widely. In addition, the scarcity of epidemiologic data about injuries occurring in day care facilities necessitates inferring, to a certain extent, that injury risks are similar to those documented for the total population of children under age five.

Nevertheless, the study does suggest the need for attention to a number of issues associated with the content, development, and monitoring of safety regulations for child care. The analysis revealed some important gaps in the regulations including inattention to playground design, firearms, and choking hazards. Given that playgrounds are the site of most reported injuries in day care centers, this oversight is especially noteworthy. Furthermore, six of the 36 standards studied were mentioned as covered under regulations other than those specific to day care by at least 20 percent of the states. It is impossible, in the context of this analysis, to assess the adequacy of those other regulations. References to other codes or standards, particularly those pertaining to fire and burn prevention, may be overlooked or misunderstood depending upon the communication and coordination among regulatory bodies or the extent to which the regulations are well-publicized. For example, knowledge about vehicle restraint laws may be more common than fire safety codes. In other instances, the failure to address an issue (e.g. firearms) may be based on the presumption that the issue is covered by other laws, a presumption that may or may not be true.

The deficiencies in the regulations reflect an inadequate regulatory process. As has been demonstrated with child automobile restraint policies, the lack of federal regulations results in diverse and inadequate state laws.[28] In the states, jurisdiction for day care safety is diffuse, with little input from injury prevention specialists. Furthermore, the relationship between regulations and the licensing process is variable among the states. Many of the regulations cannot be assessed until after a facility opens (e.g. supervision practices). Consequently, the role of inspection and relicensure may have more significance than initial licensing procedures and could benefit from further study. Interestingly, none of the national guidelines used as the basis of this analysis addressed the issue of inspection processes; however, the AAP/APHA guidelines[26] do include recommendations about inspections.

With large numbers of children in out-of-home-care other than day care centers, specific consideration of safety in family day care homes is essential. This requires attention to the delicate balance between access to affordable care on the one hand and, on the other, to the different hazards such as firearms that may be more prevalent in the home environment.

In the absence of a federal commitment, safety in child day care will remain dependent upon state actions. These efforts may be facilitated by the performance standards produced by the American Academy of Pediatrics and the American Public Health Association.[26] However helpful, they should not substitute for carefully constructed policy initiatives, preferably at the federal level, based on sound injury research. ☐

Acknowledgments

This work was supported by a grant from the Centers for Disease Control to the University of North Carolina Injury Prevention Research Center (CCR40222-04). The authors acknowledge the conceptual, editorial, and/or technical assistance of: Richard Clifford, Thomas Cole, Jo Anne Earp, Thelma Harms, Janet Heath, Deborah Lambert, Mary Linzer, Jill Moore, Eric Rodgman, Meredith Partridge, Elizabeth Sand and Mary Thompson, as well as the participation of the day care licensing personnel in the states.

An earlier version of this paper was presented at the 117th Meeting of the American Public Health Association, Chicago, Illinois, October 1989.

References

1. US Department of Commerce, Bureau of the Census: Who's Minding the Kids? Child Care Arrangements. Current Population Reports, Series P-70, No. 9. Winter 1984–1985. Washington, DC: US Govt Printing Office.
2. Gallagher SS, Finison K, Guyer B, Goodenough S: The incidence of injuries among 87,000 Massachusetts children and adolescents: Results of the 1980–81 statewide childhood injury prevention program surveillance system. *Am J Public Health.* 1984;74:1340–1347.
3. Runyan CW, Kotch JB, Margolis LH, Buescher PA: Childhood injuries in North Carolina: A statewide analysis of hospitalizations and deaths. *Am J Public Health.* 1985;75:1429–1432.
4. Waller AE, Baker SP, Szoka A: Childhood injury deaths: National analysis and geographic variations. *Am J Public Health.* 1989;79:310–315.
5. American Association of Poison Control Centers, 1988 Annual Report: National Data Collection System. *Am J Emerg Med.* 1989;7:495–545.
6. Agency for Toxic Substances and Disease Registry: The Nature and Extent of Lead Poisoning in Children in the United States. A Report to Congress. Atlanta, GA: DHHS, PHS, July 1988.
7. Baker SP, Fisher RS: Childhood asphyxiation by choking or suffocation. *JAMA.* 1980;244:1343–1346.
8. Harris CS, Baker SP, Smith GA, Harris RM: Childhood asphyxiation by food: A national analysis and overview. *JAMA.* 1984;251:2231–2235.
9. Baker SP, O'Neill B, Karpf RS: The Injury Fact Book. Lexington, MA: Lexington Books, 1984.
10. Massachusetts Department of Public Health: Statewide Comprehensive Injury Prevention Program. Safe daycare: A teacher's guide for creating safe environments for preschool children. Boston, MA: Massachusetts DPH, 1986.
11. Chatterjee BF, Barancik JI, Fratianne RB, Waltz RC, Fife D: Northeastern Ohio trauma study: V. Burn injury. *J Trauma.* 1986;26:844–847.
12. Sacks JJ, Holt KW, Holmgreen P, Colwell LS, Brown JM: Playground hazards in Atlanta child care centers. *Am J Public Health.* 1990;80:986–987.
13. Christoffel K, Christoffel T: Handguns as a pediatric problem. *Pediatric Emerg Care.* 1986;2:75–81.
14. Wintemute G, Teret S, Kraus J, Wright M, Bradfield G: When children shoot children—88 unintended deaths in California. *JAMA.* 1987;257:3107–3109.
15. Sacks JJ, Smith JD, Kaplan KM, Lambert DA, Sattin RW, Sikes RK: The epidemiology of injuries in Atlanta day-care centers. *JAMA.* 1989;262:1641–1645.
16. Personal communication, Major Douglas Hart, Public Affairs Officer, US Department of Defense, December 11, 1990.
17. Personal communication, Head Start Bureau, US Department of Health and Human Service, December 11, 1990.
18. Rivara FP, DiGuiseppi C, Thompson RS, Calonge N: Risk of injury to children less than 5 years of age in day care versus home care settings. *Pediatrics.* 1989;84:1011–1016.
19. Aronson SS: Injuries in child care. *Young Children.* 1983;38:19–20.
20. Chang AL, Lugg MM, Nebedum A: Injuries among preschool children enrolled in day care centers. *Pediatrics.* 1989;83:272–277.
21. Elardo R, Solomons HC, Snider BC: An analysis of accidents at a day care center. *Am J Orthopsychiatry.* 1987;57:60–65.
22. Landman PF, Landman GB: Accidental injuries in children in day care centers. *Am J Dis Child.* 1987;141:292–293.
23. Garrard J, Leland N, Smith DK: Epidemiology of human bites to children in a day care center. *Am J Dis Child.* 1988;142:643–650.
24. Deitch SR (ed): Health in Day Care: A

Manual for Health Professionals. Elk Grove Village, IL: American Academy of Pediatrics, 1987.
25. Bredekamp S (ed): Guide to Accreditation. Washington, DC: National Association for the Education of Young Children, 1985.
26. AAP/APHA National Health and Safety Performance Standards for Out-of-Home Child Care Programs. Draft #2. Washington, DC: American Public Health Association, March 1990.
27. US Department of Health and Human Services, Office of Human Development Services, Administration for Children, Youth and Families, Head Start Bureau: Head Start Performance Standards (45-CFR 1304). DHHS Pub. No. (OHDS) 84-31131, Washington, DC, November 1984.
28. Teret SP, Jones AS, Williams AF, Wells JK: Child restraint laws: An analysis of gaps of coverage. *Am J Public Health.* 1986;76:31–34.

Women Who Are Most at Risk for AIDS Perceive It Least, Say Loyola Researchers

Two Loyola University Chicago researchers have found that women who are most at risk for HIV are the least likely to perceive themselves as being at risk. They also discovered that women who are at a high risk are no more likely to be tested than those who are at a low risk.

Although other researchers had long suspected both of these facts, the Loyola team was the first to document it.

Of the 320 women surveyed, an almost equal number of minority and nonminority women indicated that they engaged in high-risk behavior. However, significantly fewer high-risk minority women agreed with statements such as "I am concerned that I will get AIDS" and "I am concerned that someone I know will get AIDS" than did high-risk nonminority women.

The research was performed last winter by Seth Kalichman, PhD, assistant professor of psychology at Loyola, and doctoral student Tricia Hunter.

"The message about the methods of AIDS transmittal clearly does not seem to be reaching high-risk minority women," said Kalichman. "Surprisingly, most of the women we surveyed had seen mass transit ads on AIDS prevention. We're hypothesizing that the ads may be lacking in cultural relevance since they did not get the desired response."

To gather information about women's perceived risk for AIDS, Kalichman and Hunter chose a unique and highly effective location. The pair approached local women of assorted ages, races, and socioeconomic classes at various public transportation sites in downtown Chicago and asked them to fill out anonymous surveys on AIDS.

"We had a cooperation rate of 93%," said Kalichman. "We didn't know what sort of reaction we would get, and the response exceeded all of our expectiations."

The Loyola researchers gave participants who completed the survey a flyer that explained the purpose of the project, gave their university telephone number for more information, and, most importantly, according to Kalichman, provided the correct answers to the survey.

Along with the debriefing sheet, the researchers handed out cards from the Chicago Board of Health which explained where the women could go to have AIDS testing done.

Kalichman and Hunter attribute a large part of their success to the fact that they approached the women during nonrush hour periods when they were simply waiting for public transportation to arrive. But more than once, women passed up a ride to finish the Loyola-funded survey—proof, the researchers think, of the women's own interest in the subject.

Because so many men asked to participate in the survey (which was restricted to women only), Loyola hopes to follow up with a second survey which will collect information about men's perceived AIDS risk.

Promoting Physical Activity and a Healthful Diet among Children: Results of a School-Based Intervention Study

ABSTRACT

Background: National health objectives call for improved diet and more regular physical activity among children. We tested the effects of a school-based program to improve students' diet and physical activity behavior at school.

Methods: Two of the four elementary schools in one Texas school district were assigned to intervention and two to control conditions. The three intervention components were classroom health education, vigorous physical education, and lower fat, lower sodium school lunches. Nutrients from school lunches and the total day and the amount of physical activity students obtained during physical education were assessed as outcome.

Results: Analysis of school lunches showed declines from base line to posttest in the two intervention schools of 15.5% and 10.4% for total fat, 31.7% and 18.8% for saturated fat, and 40.2% and 53.6% for sodium; posttest values were lower in the intervention schools. Observation of physical activity during physical education classes indicated an increase in the intervention schools from baseline to posttest in the percent of time children engaged in moderate-to-vigorous physical activity from less than 10% of class time at baseline to about 40% of class time at posttest; posttest values were higher in the intervention schools than in the control schools.

Conclusions: This efficacy study demonstrates the feasibility of substantially modifying school lunches and school physical education to improve children's diet and physical activity behavior at school. (*Am J Public Health.* 1991;81:986–991)

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Introduction

Childhood diet and physical activity may lead to increased risk for cardiovascular disease (CVD) in adulthood.[1] Promoting a healthful diet and regular vigorous physical activity among children is a national public health objective.[2] School is an ideal setting in which to promote healthful diet and physical activity because these topics usually are part of the health education curriculum, and federally supported food services and state-mandated physical education (PE) are widespread.[3] School food services provide nutritious meals for 73.4% of all US public school students,[4,5] but the high fat and sodium content is a concern.[6] An estimated 97% of first- through fourth-grade children in US public schools are enrolled in school PE programs for an average of 100 minutes per week.[7] However, the frequency and duration of PE classes and the amount of moderate to vigorous physical activity (MVPA) children obtain during PE may be less than recommended.[8,9]

This study was designed to influence the school environment in terms of affect on student diet and physical activity at school. The Go For Health (GFH) program included a behaviorally-based health education curriculum, fitness-oriented PE, and lower fat and sodium school lunches. We report the effects of the program on children's dietary intake and physical activity during school.

Methods

Design and Study Population

The Texas City Independent School District (TCISD) was selected for the study because it is conveniently close to the university, it has an ethnic mix of students, and it has curriculum, school lunch, and PE components. The district participated in the US Department of Agriculture school lunch program, and each school had its own on-premises kitchen, cafeteria, and food preparation staff. Third and fourth graders had PE daily, taught by PE specialists. Facilities at each school include a modern gymnasium and large outdoor playground area. The population of students was 62.3% Anglo-American, 20.9% Mexican-American, and 14.8% Afro-American. The ethnic distributions were comparable for both treatment and control conditions.

Two of the four elementary schools (kindergarten through fourth grade) were assigned to intervention and two to control conditions. Third and fourth graders were assessed annually during three spring data collection periods.

Intervention

The three GFH intervention components, based on social cognitive theory,[10] were the Go For Health Curriculum, Children's Active Physical Education (CAPE), and the New School Lunch (NSL).[11] Following the model of Charter

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This paper was submitted to the journal September 13, 1990, and accepted with revisions April 23, 1991.

TABLE 1—Average Nutrients per Lunch (N = 12 Meals per School) at Baseline, Midtest, and Posttest, with 95% Confidence Intervals: Go for Health Recipe Analysis

Treatment condition	Energy (kcal)	Protein (g)	Carbohydrate (g)	Fat (g)	Cholesterol (mg)	Vitamin A (IU)	Calcium (mg)
Control school 1							
Baseline[a]							
Mean	1049.2	38.3	115.1	48.4	123.8	2722.7	535.5
Lower 95% CI	1014.3	37.4	112.7	46.0	116.5	2291.0	516.4
Upper 95% CI	1084.1	39.2	117.3	50.9	131.1	3154.4	554.6
Midtest							
Mean	997.5	36.2	107.7	46.9	113.2	2985.3	537.2
Lower 95% CI	949.3	35.2	104.5	43.4	104.5	2301.4	510.5
Upper 95% CI	1045.8	37.3	111.0	50.3	121.8	3669.2	564.0
Posttest							
Mean	898.2	35.3	100.4	39.5	94.6	2071.5	480.3
Lower 95% CI	870.4	34.4	98.2	37.8	90.4	1738.3	461.4
Upper 95% CI	926.0	36.2	102.4	41.3	98.7	2404.7	499.3
Control school 2							
Baseline[a]							
Mean	1049.2	38.3	115.1	48.4	123.8	2722.7	535.5
Lower 95% CI	1014.3	37.4	112.7	46.0	116.5	2291.0	516.4
Upper 95% CI	1084.1	39.2	117.1	50.9	131.1	3154.4	554.6
Midtest							
Mean	1038.3	37.1	114.9	47.8	112.2	2418.6	539.9
Lower 95% CI	996.4	35.7	112.4	44.9	104.2	2019.6	507.9
Upper 95% CI	1080.3	38.4	117.8	50.6	120.2	2817.7	572.0
Posttest							
Mean	943.5	37.1	103.6	42.3	111.7	2106.0	511.7
Lower 95% CI	918.6	36.1	102.2	40.6	103.4	1756.4	488.7
Upper 95% CI	968.4	38.0	105.3	43.9	119.9	2455.5	534.7
Intervention school 3							
Baseline							
Mean	1077.5	37.3	117.8	50.8	137.7	2809.4	544.0
Lower 95% CI	1037.8	36.3	114.9	48.1	129.8	2365.6	522.3
Upper 95% CI	1117.1	38.3	120.6	53.5	145.6	3253.1	565.6
Midtest							
Mean	890.4	37.7	105.3	35.4	98.1	3559.6	514.5
Lower 95% CI	856.3	36.4	102.5	33.4	91.6	3020.8	496.2
Upper 95% CI	924.5	38.9	108.1	37.4	104.7	4098.4	532.7
Posttest							
Mean	849.3	35.0	100.8	34.0	79.0	3393.0	474.7
Lower 95% CI	816.8	34.4	97.8	32.0	76.8	2916.7	462.1
Upper 95% CI	881.8	35.7	104.0	35.9	81.1	3869.3	487.2
Intervention school 4							
Baseline							
Mean	1021.0	39.3	112.2	46.1	110.0	2636.0	527.0
Lower 95% CI	856.6	37.9	110.4	43.6	101.8	2210.9	508.3
Upper 95% CI	985.4	40.6	114.4	48.5	118.1	3061.0	545.7
Midtest							
Mean	887.3	33.8	108.2	35.5	83.5	3430.8	501.5
Lower 95% CI	856.6	32.7	106.1	33.5	78.2	2903.0	481.3
Upper 95% CI	918.0	34.8	110.3	37.5	88.7	3958.6	521.7
Posttest							
Mean	840.9	35.7	96.9	34.5	79.3	3907.6	469.5
Lower 95% CI	800.7	35.0	93.0	32.1	77.1	3305.2	452.4
Upper 95% CI	881.1	36.4	100.6	37.0	81.4	4509.9	486.6

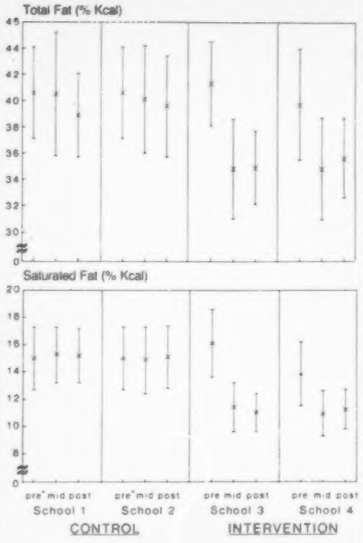
[a] Baseline for control schools imputed from the average of the intervention schools.

and Jones,[12] we employed a four-step approach to program implementation and institutionalization.[13] To foster quality implementation, the GFH staff provided ample training, consultation, and technical support to both teachers and food service staff.

The purposes of the classroom health education, which consisted of six, behaviorally based modules, were to teach knowledge and skills essential to lifelong performance of the target diet and physical activity behaviors and foster the transfer of learning about these behaviors from school to out-of-school environments.[10]

The NSL was designed to provide lower-fat, lower-sodium lunches within the context of the existing school lunch program. Food purchasing, menus, recipes, and food preparation practices were modified.

CAPE, which consisted of five, 6- to 8-week units, was designed to encourage enjoyable MVPA among children during PE classes. Each unit included two or three main cardiovascular fitness activities, such as dancing, running, aerobic games, jump rope, and obstacle courses. Each class session consisted of warmup, fitness development, cool down, and skill development or game activities.



*Imputed values—based on average of intervention schools

FIGURE 1—Means and 95% Confidence Intervals for Total Fat and Saturated Fat as a Percent of Calories in School Lunches (N = 12 meals per school) in Control and Intervention Schools at Baseline (Pre), after 1 Year (Mid), and after 2 Years (Post).

Measures and Procedures

Program effects were assessed by analyzing the nutrient contents of lunches, conducting 24-hour dietary recalls, and observing students' physical activity during PE.

Nutrient Analysis of School Lunches. The identical menu was repeated in March and April each year of the study. At baseline, 12 meals were selected randomly from the March and April menus and analyzed for nutrient content. Recipe analyses, based on detailed interviews with each cook in the intervention schools at baseline and in all four schools at midtest and posttest, were conducted by trained staff nutritionists and analyzed by the Nutrition Coding Center (NCC).[14]

Diet Recall. The 24-hour dietary recall interview was administered at posttest to random, cross-sectional subsamples of children, stratified by study school. Interviewers were trained according to NCC procedures and followed the NCC protocol for diet interviews. Each child was interviewed at home in the company of one parent, who was asked about ingredients, food preparation, and other items as needed.

Physical Activity. The time (in minutes) of MVPA performed by children during physical education was assessed by trained observers using the Children's Physical Activity Observation Form (CPAOF).[15] Study schools were observed on randomly selected days over a 2-month period each spring; all third- and fourth-grade classes were observed. Observers recorded on a minute-by-minute basis the type (e.g., basketball, jump rope) and intensity of physical activity. The CPAOF intensity categories have been validated against heart rate,[15] and the average heart rate for MVPA was 172.2 (SD = 21.2) beats per minute. Interobserver agreement was 97.0% (n = 57 paired observations).

Analysis

For energy and selected nutrients of potential concern in this age group, we calculated the means and 95% confidence intervals (CIs) of the repeated samples of school lunches in each school. For each separate treatment condition, we calculated the mean nutrient intakes from the 24-hour dietary interviews for selected nutrients and examined the contribution of school and bag lunches to total daily nutrient intakes.

For each school at each reporting period, we calculated the MVPA means, standard deviations, medians, and 95% CIs for the medians.[16] The median is the most appropriate statistic for purposes of comparing treatment effects because increases in the amount of MVPA on the part of only a few students could affect the mean value, obscuring true program effects. Percent of class time is preferable to minutes as an outcome because the scheduled length of PE classes varied somewhat from year to year, and the effective class time available for PE teachers and students, although similar in each school for a given year, varied somewhat from school to school due to local scheduling. The annual surveys are presented separately for each grade because most of one year's third graders became the next year's fourth graders; hence the data for each grade are not independent across years.

Results

Diet

Recipe Analysis. Table 1 includes the means and the 95% CIs for total energy (in kilocalories [1 kcal = 4.2 kJ]) and six nutrients in the 12 meals served in each study school each year. Baseline values for the control schools, which were not collected directly, were estimated by averaging the values of the two intervention schools. Declines from baseline to posttest in each school for protein, carbohydrates, total fat, cholesterol, and calcium are the product of declines in energy. The decline in calories served in the intervention schools appears to have been due mainly to a reduction in the amount of fat and carbohydrates. Despite the declines in energy, vitamin A and calcium did not decline, and posttest values for all schools exceeded the USDA school lunch requirements for each nutrient. For example, at every reporting the average intake of energy in each school exceeded 33% of the total daily requirement for energy, which is 600 to 700 kcal per meal.[17]

Shown in Figure 1 for each school is the average total fat and saturated fat as a percent of kcal served in the sample of 12 lunches. For both intervention schools the upper CIs of the posttest means were less than the pretest means, and in school 3 the upper and lower CI values did not overlap. The mean percent fat declined in school 3 by 15.5%, from 41.3% to 35.6% of calories, and in school 4 by 10.3%, from 39.7% to 35.6% of calories. Average percent saturated fat declined in school 3 by 31.7%, from 16.1% to 11.0% of calories, and in school 4 by 18.8%, from 13.8% to 11.0% of calories. The means for percent total fat and saturated fat at posttest in the control school were within the imputed pretest 95% CIs.

Figure 2 shows that the sodium content in the school food declined from baseline to posttest in school 3 by 53.6% from 2141.1 mg to 993.0 mg, and in school 4 by 40.2%, from 1730.0 mg to 1034.5 mg. These differences remained after controlling for kcals served. Sodium in the control schools did not decline from the imputed baseline value.

Dietary Recall. Table 2 shows the analysis of the posttest 24-hour recalls for the nutrients of interest. Students in the intervention group, compared with students in the control group, reported fewer calories (2094 vs 2135 [1 calorie = 4.2 J]) and less total fat (77 g vs 90 g), saturated fat (29 g vs 34 g), and sodium (2873 mg vs 3378 mg) for the total day. In comparison with control group students, intervention group students reported consuming less total fat, saturated fat, and sodium in both tray and bag lunches.

Physical Activity

Table 3 shows medians, means, and standard deviations for minutes of MVPA for observed students in grades three and four at each study school at baseline, midtest, and posttest. At baseline, mean MVPA for third and fourth graders was less than 3 minutes at each school; medians were 2.0 minutes or lower. At posttest, mean minutes had increased in school 3 to 11.7 (third grade) and 15.0 (fourth grade) and in school 4 to 16.2 (third grade) and 16.1 (fourth grade) per class.

Median minutes as a percent of class time and 95% CIs around the medians are shown for third and fourth grade in Figure 3. For both grades, the median percent of class time in MVPA at baseline was zero in three schools and less than 10% in school 3. At posttest, the control school medians were still no greater than 5% of class time. In the intervention schools, however, the posttest medians were substantially higher, 28% of class time for third graders in school 3, and 39% of class time or greater for the other intervention groups. Except for fourth grade in school 3, the upper and lower 95% CI values do not overlap with the baseline means.

Discussion

The results show strong positive program effects on children's diet and physical activity at school. At posttest, school lunches in the intervention schools provided less energy, fat, and sodium compared with baseline and with the control schools, while adequate levels of other essential nutrients were maintained. Similarly, children's physical activity in the intervention schools was much higher at posttest than at baseline and in comparison with the control schools.

Several studies have demonstrated that sodium reductions can be accomplished by intervention directed at school meals.[18,19] However, no studies have reported systematic reductions in fat and sodium in school lunches. In the GFH intervention schools, sodium content was reduced to target levels, saturated fat reductions of 31.7% and 18.8% occurred, and posttest values approached the target goal of 10% of kcals served. Total fat reductions were modest, 15.5% and 10.3%, and the target goal of 30% of kcals was not reached, indicating that it is easier, even in institutional settings, to substitute fat type than to reduce the amount of fat.

The 24-hour dietary recall data indicate that the changes in nutrients served in

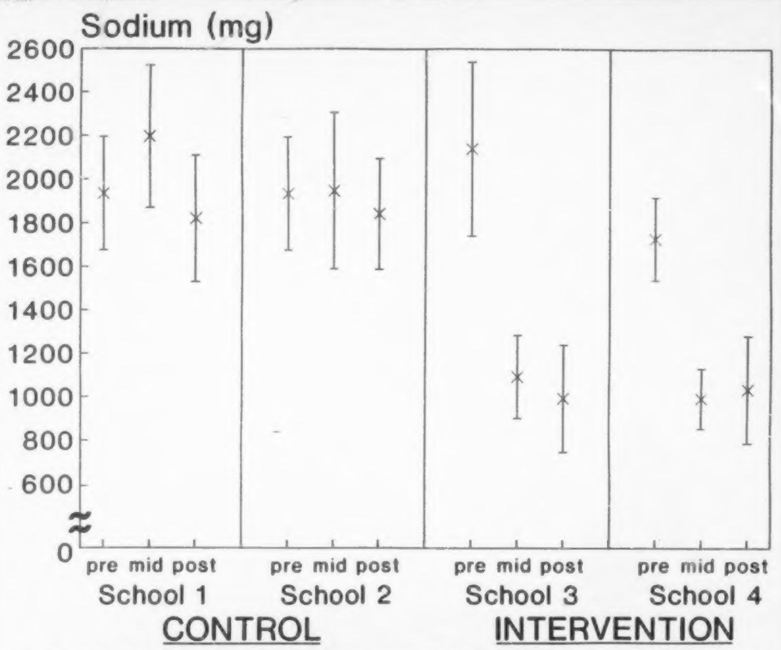


FIGURE 2—Means and 95% Confidence Intervals for Sodium in School Lunches (N = 12 Meals per School) in Control and Intervention Schools at Baseline (Pre), after 1 Year (Mid), and after 2 Years (Post).

TABLE 2—Means and 95% Confidence Intervals of Selected Nutrients from Bag or Tray Lunch Based on 24-Hour Diet Recalls in the Intervention and Control Schools

Variables	Energy (kcal)	Total fat (g)	Saturated fat (g)	Sodium (mg)
		Control		
Total day (n = 68)				
Mean	2135.3	90.0	33.9	3377.6
Lower 95% CI	1978.3	80.8	30.3	3044.1
Upper 95% CI	2292.3	99.1	37.6	3711.0
Tray lunch (n = 49)				
Mean	806.9	34.8	13.8	1283.4
Lower 95% CI	730.7	30.8	11.9	1137.2
Upper 95% CI	883.0	38.8	15.8	1429.6
Total day average (%)	37.8	40.1	40.7	39.7
SE	1.8	2.4	2.4	2.3
Bag lunch (n = 19)				
Mean	681.4	27.3	9.0	966.0
Lower 95% CI	568.8	21.5	6.9	794.8
Upper 95% CI	794.1	33.0	11.0	1137.3
Total day average (%)	35.7	34.4	32.5	33.4
SE	2.6	2.7	3.4	2.7
		Intervention		
Total day (n = 67)				
Mean	2094.5	78.6	28.5	2872.6
Lower 95% CI	1922.6	70.0	25.0	2554.2
Upper 95% CI	2266.5	87.2	32.0	3190.7
Tray lunch (n = 48)				
Mean	790.2	29.7	10.0	909.1
Lower 95% CI	676.9	25.6	8.3	756.6
Upper 95% CI	903.4	33.8	11.6	1062.7
Total day average (%)	36.5	38.0	35.4	32.5
SE	1.9	2.2	2.3	2.0
Bag lunch (n = 19)				
Mean	657.7	25.1	7.8	789.5
Lower 95% CI	516.8	18.8	5.6	645.5
Upper 95% CI	798.6	31.3	10.0	933.5
Total day average (%)	36.1	37.2	32.6	31.3
SE	2.7	2.8	3.2	2.4

TABLE 3—Average Time (in Minutes) of MVPA Observed during Physical Education in Control and Intervention Schools

Treatment Condition	Third Grade N	Third Grade Median	Third Grade Mean	Third Grade SD	Fourth Grade N	Fourth Grade Median	Fourth Grade Mean	Fourth Grade SD
Control								
School 1								
Baseline	32	0.0	0.4	0.9	41	0.0	0.3	0.7
Midpoint	27	1.0	2.8	5.0	36	0.5	1.1	1.5
Posttest	37	2.0	2.9	3.5	35	1.0	1.5	1.9
School 2								
Baseline	34	0.0	0.5	1.1	26	0.0	0.4	0.9
Midpoint	24	1.0	2.0	2.2	10	0.0	2.2	3.4
Posttest	36	2.0	3.5	3.9	51	1.0	4.5	5.9
Intervention								
School 3								
Baseline	31	2.0	2.0	1.4	45	2.0	2.4	1.9
Midpoint	34	1.5	2.6	2.8	24	2.5	6.8	7.4
Posttest	38	10.5	11.7	7.5	26	20.0	15.0	10.6
School 4								
Baseline	32	0.0	2.8	5.0	36	0.0	1.9	3.6
Midpoint	38	4.5	4.9	4.3	24	7.0	8.0	4.4
Posttest	58	16.0	16.2	7.8	49	16.0	16.1	7.4

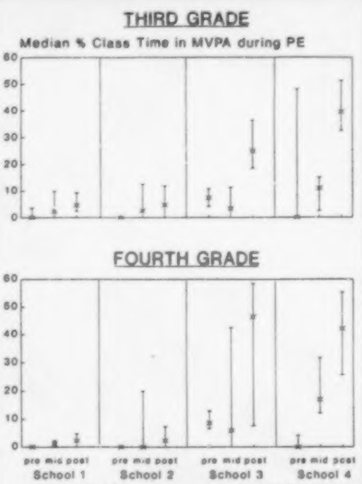


FIGURE 3—Medians and 95% Confidence Intervals for Percentage of Physical Education Class Time in Moderate to Vigorous Physical Activity (MVPA) in Control and Intervention Schools at Baseline (Pre), after 1 Year (Mid), and after 2 Years (Post); Third and Fourth Grade.

school lunch contributed to improvements in total daily dietary intake of calories, total fat, saturated fat, and sodium. Children did not make up in other meals for reductions of fat and sodium in school lunches. Energy, calcium, and vitamin A remained adequate at posttest in all schools, suggesting that reductions in fat can be accomplished without undue reductions in other essential nutrients.

Several studies have demonstrated that it is possible to modify PE to improve cardiorespiratory fitness.[20-22] Our results indicate that it is possible to increase time devoted to MVPA from less than 10% to more than 40% of available class time. At posttest, the typical student in the intervention schools obtained about 16 minutes of MVPA per daily PE period, or 80 minutes of MVPA per week. It must be noted that these modest results were obtained after 2 years of intervention that involved one staff person virtually full time in the provision of intensive training of PE teachers. Because PE accounts for nearly half of children's total daily physical activity,[23] the extremely low level of MVPA observed in all schools at baseline is a cause for concern, particularly if future research determines that low levels of activity during PE are prevalent in American schools.

This is the first US study to demonstrate the potential of altering the school environment to promote a more healthful diet and more vigorous physical activity among children, although at least one successful European study has been reported.[24] Strengths of the present study include planned implementation of theory-based individual and organizational interventions, two intervention and two control schools within the same district, and objective measures of diet and physical activity. Limitations of the study include the lack of baseline recipe data for the control schools, nonrandom assignment of schools to treatment conditions, and the small number of study schools.

The results of this efficacy study suggest that school is a potentially important site for improving children's diet and physical activity and that such changes are possible. In our experience, however, schools do not change readily, and substantial staff training should accompany policy changes regarding healthful school lunch and vigorous physical education. Before widespread implementation of these changes occurs, effectiveness studies with larger numbers of schools are needed to determine the ability to generalize these approaches and to assess cost effectiveness. ☐

Acknowledgments

This research was funded by NHLBI Grant 33376. We gratefully acknowledge the administrators, teachers, and staff of the Texas City Independent School District for their support and cooperation. Also, we thank Renee Fleishman for data preparation and Iris Huang for statistical analyses.

This research was presented at the annual meeting of the American Public Health Association held in New York City September 30 to October 4, 1990.

References

1. Hetzel BS, Berenson GS. *Cardiovascular Risk Factors in Childhood: Epidemiology and Prevention.* New York: Elsevier Science Publishers; 1987.
2. Public Health Service. *Promoting Health/Preventing Disease: Year 2000 Objectives for the Nation.* Washington, DC: US Dept of Health and Human Services; 1991. DHHS Publication II (PHS) 91-50213.
3. Pigg RM Jr. The contribution of school

health programs to the broader goals of public health: the American experience. *J Sch Health.* 1989;59:25–30.

4. Radzikowski J, Gale SK. Requirements for the national evaluation of school nutrition programs. *Am J Clin Nutr.* 1984; 40(suppl):365–367.
5. Hanes S, Vermeersch J, Gale S. The national evaluation of school nutrition programs: program impact on dietary intake. *Am J Clin Nutr.* 1984;40(suppl):390–413.
6. Frank G. Primary prevention in the school arena: a dietary approach. *Health Values.* 1983;7:14–21.
7. Ross JG, Pate RR, Corbin CB, Delpy LA, Gold RS. The National Children and Youth Fitness Study II: What is going on in the elementary physical education program? *J Phys Ed Rec Dance.* 1987;58:78–84.
8. National Association for Sport and Physical Education. NASPE 1988: a year in review. *NASPE News.* 1989;24:1.
9. Simons-Morton BG, Parcel GS, O'Hara NM, Blair SN, Pate RR. Health related physical fitness in childhood: status and recommendations. *Annu Rev Public Health.* 1988;9:403–425.
10. Parcel GS, Simons-Morton BG, O'Hara NM, Baranowski T, Wilson B. School promotion of healthful diet and physical activity: impact on learning outcomes and self-reported behavior. *Health Educ Q.* 1989;16:181–199.
11. Bandura A. *Social Foundations of Thought and Action.* Englewood Cliffs, NJ: Prentice-Hall, Inc; 1986.
12. Charter WW, Jones JE. On the risk of appraising non-events in program evaluation. *Educ Res.* 1973;2:5–7.
13. Parcel GS, Simons-Morton BG, Kolbe LJ. School health promotion: integrating organizational change and student learning strategies. *Health Educ Q.* 1988;15:435–450.
14. Dennis B, Ernst N, Hjortland N, Tillotson J, Grambsch VB. The NHLBI nutrition data system. *J Am Diet Assoc.* 1980;77: 641–645.
15. O'Hara N, Baranowski T, Simons-Morton BG, Wilson B, Parcel GS. Validity of the observation of children's physical activity. *Res Q Exercise Sport.* 1989;60:42–47.
16. Walsh JE. Nonparametric confidence intervals and tolerance regions. *In:* Sarhan AE, Greenberg BG, eds. *Contributions to Order Statistics.* New York: John Wiley; 1962:136–143.
17. National Academy of Sciences: Recommended Dietary Allowances, 10th ed. Washington, DC: National Academy of Sciences; 1989.
18. Frank G, White M. Application of a sodium-modified recipe program in school lunch. *Sch Food Serv Res Rev.* 1983;7:2–4.
19. Ellison RC, Capper AL, Goldberg RG, Witshi JDC, Stare FJ. The environmental component: changing school food service to promote cardiovascular health. *Health Educ Q.* 1989;16:285–297.
20. Dwyer T, Coohan W, Leitch O, et al. An investigation of the effect of daily physical activity on the health of primary school students in South Australia. *Int J Epidemiol.* 1983;12:308–313.
21. Duncan B, Boyce T, Itami R, Paffenbarger N. A controlled trial of physical fitness programs for fifth grade students. *J Sch Health.* 1983;53:467–471.
22. Coates TJ, Jeffery RW, Slinkard LA. Heart healthy eating and exercise: introducing and maintaining changes in health behaviors. *Am J Public Health.* 1981;71:15–23.
23. Simons-Morton, BG, O'Hara NM, Simons-Morton DG, Parcel GS. Children and fitness: a public health perspective. *Res Q Exercise Sport.* 1987;58:295–303.
24. Grafner L, Heinrich J, Knappe J, Holtz H. Atherosclerosis precursors in school children—results of a two-year intervention study. *Cor Vasa* 1987;29:421–427.

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Contact Ellen Lemberg, RN,C, MS, Nurse Practitioner Symposium Office, School of Nursing, University of Colorado Health Sciences Center, 4200 East 9th Avenue, Box C287, Denver, Colorado 80262. Tel: 303/270-7436.

Attitudes toward Abortion among Parents of Children with Cystic Fibrosis

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ABSTRACT

Background: DNA prenatal diagnosis for cyctic fibrosis (CF) has been available for parents of affected children since late 1985.

Methods: Using anonymous questionnaires, we surveyed 395 parents of children with CF at 12 New England CF centers with regard to 12 maternal or family situations and 11 fetal characteristics; 271 (68%) responded.

Results: The majority supported legal abortion in the first trimester for all 23 situations; 58% would abort for severe mental retardation (MR), 40% would abort for a genetic disorder leading to death before age five years, 41% for a child bedridden for life, 35% for moderate MR, 20% for CF and 17% for a severe incurable disorder starting at age 40 years. Few would abort for a disorder starting at age 60 years, for genetic susceptibility to alcoholism or for sex selection. Variables most strongly related to abortion for CF were attitudes of spouse, respondent's siblings, and CF doctor toward abortion for CF as well as infrequent attendance at religious services.

Conclusions: Prenatal diagnosis may not reduce substantially the number of CF births to parents of CF children because most do not accept abortion for CF. (*Am J Public Health.* 1991;81:992–996)

Introduction

Parents of children with genetic disorders are frequently ambivalent toward selective abortion of affected fetuses and may regard the abortion as a rejection of their affected child. Although some studies report that the majority of these parents do not consider abortion acceptable for the disorder in question,[1–8] other studies indicate that such parents may be more willing to abort than the general population.[9–10]

Attitudes toward abortion follow a spectrum of perceived severity, severe mental retardation (MR) being the most crucial factor. Most women (94% to 97%) with prenatally diagnosed fetal abnormalities involving severe MR (e.g., trisomies 13, 18, or 21) choose abortion.[11] Fewer women (62%) choose abortion for sex chromosome abnormalities (e.g., 45, X Turner syndrome or 47, XXY Klinefelter syndrome) that are not ordinarily associated with retardation.[12] Severe physical disability ranks next. Among women undergoing MSAFP (maternal serum α-fetoprotein) screening for neural tube defects, 57% would abort if there "was a very good chance that the child would be born with a serious physical handicap [sic], for example, blindness or paralyzed legs."[13]

Some common genetic disorders such as cystic fibrosis (CF) involve neither MR nor significant physical limitations, but carry a reduced life expectancy and increased morbidity. CF is the most common autosomal recessive disorder affecting Caucasian populations, with an incidence of 1 in 2000 to 3000 births. About 30 000 individuals in the United States are affected. Improved palliative treatment has extended life expectancy and improved quality of life. In 1988, 75% survived into their late teens, half reached the age of 26, and about 40% reached the age of 30, though few survived to their fifth decade.[14]

Since late 1985, identification of deoxyribonucleic acid (DNA) probes linked to the CF gene has allowed accurate prenatal diagnosis for families who have had a previous child with CF and for whom specimens for the affected child and both parents can be analyzed in parallel with amniotic fluid or chorionic villi (CVS) specimens.[15–16] In other nations the majority of such families (65% in Belgium,[17] 52% in Wales[18]) would abort a CF fetus. In France, parents chose to terminate 53 of 56 CF fetuses following prenatal diagnosis.[19] There are no comparable studies in the United States. Our specific aims were (1) to find out how parents of affected children viewed abortion for CF, and (2) to see where parents placed CF in a wider spectrum of situations that might justify abortion.

Methods

Questionnaire Content

Our questions on abortion were part of a larger study of psychosocial factors

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This paper was submitted to the journal October 2, 1990, and accepted with revisions April 4, 1991.

affecting use of prenatal diagnosis for CF in six New England states. We anticipated that parents might be less likely to abort for CF than for severe MR, severe physical disability, conditions that threatened the mother's life or health, and rape or incest, and more willing to abort for CF than for late-onset or cosmetic conditions.

We asked about attitudes toward abortion in 23 situations, including 12 maternal or family situations and 11 conditions affecting the child. Respondents were asked to give one of three responses: (1) "I would have an abortion," (2) "I would not have an abortion, but it should not be prohibited for others," or (3) "I think abortion should be prohibited by law." We included situations described in opinion polls (mother's life, rape, incest), or used as rationales for abortion (maternal age under 15, mother's career, financial burden, family completed). In describing fetal characteristics, we avoided listing names of specific disorders and instead briefly described the child's condition (e.g., instead of trisomy 18, "severe mental retardation: child unable to speak or understand"; instead of Tay-Sachs, "severe genetic disorder leading to death before age 5"; instead of Huntington disease, "severe painful disorder starting at age 40, incurable"). We included several conditions that are not presently diagnosable prenatally, such as susceptibility to alcoholism, "severe incurable disorder at age 60" (Alzheimer disease), and "severe, untreatable obesity." We included obesity because of concerns that, if ever given the opportunity, some parents may make prenatal selections on cosmetic grounds, such as hair, eye, or skin color, or stature.[20] Finally, we included sex selection because, despite much publicity and two recent surveys of physicians,[21-23] there are no surveys of parental attitudes.

Previous studies have found associations between abortion attitudes and religion,[9,10,24] religiosity,[13,25] education,[10,25,26] income,[10] previous elective abortion,[9] and interpretation of risk,[9] but a recent study of CF families in the United Kingdom found no association between abortion attitudes and social class.[18] The 15-page questionnaire included the above variables and sections on reproductive history, reproductive plans, health of the affected child, expectations about the child's activities 5 and 10 years from now, knowledge about new genetic tests, and standard sociodemographic data. Most questions called for categorical (yes-no) responses. Five-point Likert scales were used to describe the interpretation of risk and the attitudes of spouses, members of extended family, and medical professionals toward abortion for CF, and (in a separate question) support of those persons carrying a CF fetus to term.

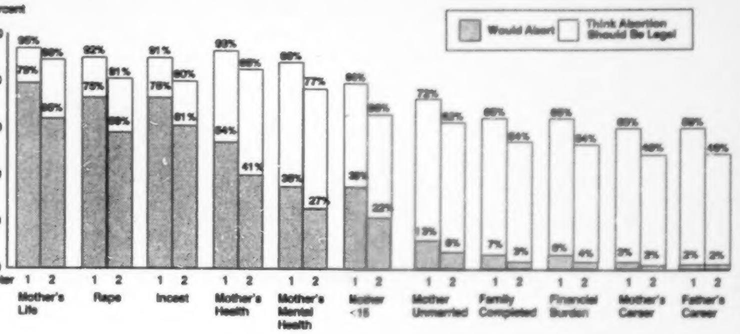


FIGURE 1—Abortion for Maternal or Family Situation (N = 271).

There are liabilities in studying attitudes rather than actual behaviors. Low prevalence and the practical difficulties of obtaining abortion histories limited us to a study of attitudes. Some studies have found attitudes strongly related to subsequent behavior.[24,27]

Distribution

Starting in January 1989, all 12 CF centers in New England distributed the questionnaires to parents of children enrolled as patients. Depending on the clinic, questionnaires were either given out at regular clinic visits over a 4-month period (most CF patients visit every 3 months) to be returned by mail, or were mailed directly to families. The two modes of administration produced almost identical response rates. In order to ensure anonymity, all questionnaires were returned directly to project staff at the Shriver Center, not to the clinics.

Data Analysis

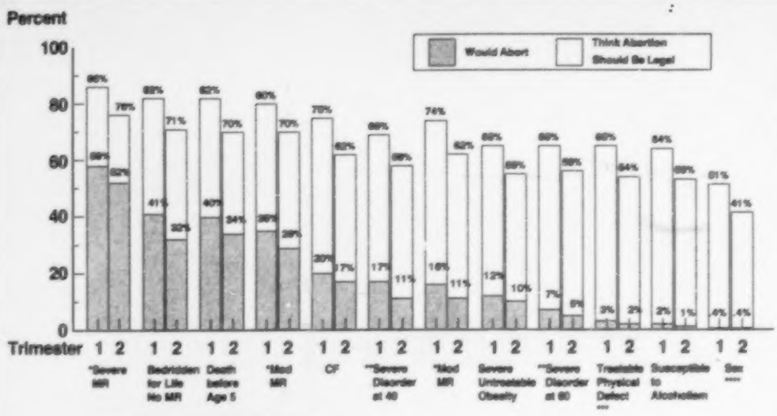
Questionnaire data were entered into Statistical Package for the Social Sciences (SPSSX) and Statistical Analysis System (SAS) programs. Using the statement "I would have an abortion" in the first trimester as the dependent variable, we looked for associations with sociodemographic variables, child's health, future expectations, interpretation of risk, and extended family's attitudes toward abortion. The five-point Likert scales were reduced to three points in order to eliminate small cells for "strongly approve." Analyses of subsets by gender, religion, and religiosity (weekly attendance) were conducted to rule out the possibility of interaction.

For each of the 23 abortion questions, all variables that associated, at the zero-order level, with "I would have an abortion" were entered into a stepwise logistic regression. This method orders each independent variable in terms of its strength of association with the dependent variable, while controlling for other variables. For each independent variable that was significantly associated, the method provided an odds ratio for personal acceptance of abortion.

Profile of Respondents

Of 395 parents asked to participate, 271 (68%, 228 families) responded. Their children's health status corresponded generally with the national clinical profile of CF.[11,28-29] The children's median age was seven; 90% had pancreatic involvement; 73% required chest physical therapy, including 46% who required it from one to five times daily; and 42% had been hospitalized within the last year.

Parents appeared typical of CF families in other studies: median age was 35; 36% were college graduates and median income was $30 000 to $40 000. These figures were comparable to an earlier California study of CF families' attitudes toward prenatal diagnosis.[30] Seventy-eight percent were living with the affected child's other parent, and 57% were Catholic, figures comparable to a New York City study.[31] The high proportion of Catholics may result from greater frequencies of the CF gene in the Irish, Italian, and French populations found in New England.[14,31] Of respondents, 73% were fe-



*Severe MR: Unable to speak or understand
Moderate MR: Could communicate, not live independently
Mild MR: Could hold job and live independently
**Develop severe painful disorder, incurable
***Cleft palate, nearsightedness
****Child is not sex desired by parents

FIGURE 2—Abortion for Fetal Characteristics (N = 271).

male and 13% had had a previous elective abortion. In all, 52% had been surgically sterilized (compared with 62% in a previous study[30]), 15% were either beyond reproductive age or no longer living with the child's other parent, and 33% were still fertile and at risk for having another child with CF.

Although 27 persons did not respond to the question about abortion for CF in the first trimester or to other questions about abortion, there were no statistically significant differences between respondents and nonrespondents.

Results

Responses are described in Figure 1 (maternal or family situations) and Figure 2 (fetal characteristics). The majority believed that abortion should be legal in all 23 situations during the first trimester and in 20 of the 23 situations during the second trimester (white bars). For all situations, fewer respondents approved legal abortion in the second trimester than in the first trimester, with an average difference of 11 percentage points.

There was a steady gradation in personal acceptance of abortion (shaded bars), running from "mother's life in danger" down to "child would interfere with father's career" (Figure 1) and from "severe mental retardation" to "child is not of sex desired by parents" (Figure 2). Severe MR was the only fetal characteristic for which the majority (58%) would themselves abort (Figure 2). For CF itself, 20% would abort in the first trimester, and 17% in the second trimester.

In both Figures 1 and 2, 80% to 100% of those represented in a shaded bar are also represented in the shaded bars to the left. For example, of those who would abort for CF, 86% would also abort for moderate MR, 88% if the child would be bedridden for life, and 100% if the child would die before age five or have severe MR. An average of 9% fewer women would abort in the second trimester than in the first trimester for maternal or family situations, and an average of 5% fewer women would abort in the second trimester for fetal characteristics. Men made less differentiation between trimesters (6% for maternal situations and 1% for fetal characteristics).

Characteristics associated at the zero-order level with personal willingness to abort are presented in Table 1. Men were more likely than women to say either that they would abort or that abortion should be prohibited by law. Men eschewed the middle ground of not aborting but permitting it for others. Gender was not related to any responses concerning fetal characteristics.

For all maternal or family situations and all fetal characteristics except sex selection, attitudes toward abortion were associated with respondent's perception of attitudes of spouse, mother, father, and siblings toward abortion of a fetus with CF. The majority thought that their spouses (65%), mothers (60%), fathers (55%), and siblings (51%) would disapprove. These views were closely interrelated, with Pearson correlation coefficients of >.6. Family attitudes toward abortion for other disorders were not surveyed.

In addition, the following variables (not reported in Table 1) were associated with abortion for CF: perceived approval of abortion for CF by father-in-law or mother-in-law, approval of abortion for CF by affected child or by other children, approval of abortion for CF by CF doctor or genetic counselor, and perceived absence of support from spouse, family doctor, or CF doctor for carrying a fetus with CF to term.

Not associated with any attitudes toward abortion, including abortion for CF, were age, fertility status, marital status, number of children, CF child's health, future expectations about the CF child, cost of the child's illness, knowledge about prenatal diagnosis, interpretation of genetic risk, and reproductive intentions. No sociodemographic variables were significantly related to abortion for obesity, severe disorder at 60, susceptibility to alcoholism, treatable defect, or sex selection.

Stepwise logistic regression analyses were performed for all abortion situations in Table 1. Characteristics strongly and independently related to willingness to abort ($P < .10$) are indicated.

Some general patterns emerged from the regression analyses. Religious background was related largely to maternal or family situations, where traditional Catholic social values may remain, rather than to abortion for fetal characteristics. In many situations, those with higher education were more willing to abort than those with less education.

Family attitudes were among the strongest factors related to personal abortion decisions. For persons living in a family context, it is difficult to make an abortion decision that contradicts the values of their spouses and extended families. Respondents' perceptions of their mothers' views were especially important for maternal or family situations. For spousal views, respondents' perceptions were accurate in the 43 families in which both spouses responded.

Regression results for abortion for CF and severe MR are reported in Tables 2 and 3. Table 2 shows that parents' perceptions of the views of their CF doctors toward abortion for CF were significant. In all, 15% thought their CF doctors would approve, 45% thought they would disapprove, and 40% thought they would be neutral.

TABLE 1—Psychosocial Characteristics Associated with Personal Willingness to Abort in Selected Situations (N = 271)

	Abortion Situation														
Psychosocial Characteristic	Mother's life	Rape	Mother's health	Mother's mental health	Mother under 15	Mother unmarried	Family completed	Financial burden	Severe MR	Bedridden for life	Death before 5	CF	Moderate MR	Mild MR	Severe disorder at 40
Religion (non-Catholic)	b	b			b	b	b	a				a			
Infrequent attendance at religious services	b	b	a	a	a	a	a	a	b	a	b	b	a	a	b
Higher education (years)	b	a	b	b		b	b			a	b		b	b	b
Higher income	a	b	a	a	b	b	a	a		b	a	a	a	a	a
Occupation (professional)									a	a	a	a	a	a	
Respondent's gender															
Female				b	a										
Male	a		a			a	a								
Previous elective abortion	b						a	a	b	b	a	a	b	b	
Approval of abortion for CF by spouse	a	a	a	a	a	b	b	a	b	a	a	b	a	b	b
Respondent's siblings	a	a	a	a	a	a	a	a	a	a	a	b	b	a	a
Respondent's mother	b	b	b	b	b	a	a	a	b	b	b	a	b	b	b
Respondent's father	a	a	a	a	b	a	a	a	a	a	a	a	a	a	a

Note: a = associated at zero-order level, $P < .05$; b = associated in stepwise logistic regression, $P < .10$.

Discussion

The major finding, that a minority of parents of children with CF (20%) would abort a CF fetus, suggests that prenatal diagnosis will not lead to a substantial reduction in the number of CF births among this group, at least in the United States. Most parents in this sample apparently do not regard selective abortion for CF as an acceptable reproductive option, although they believe this option should be available to others. Many affected families may not welcome public health efforts to provide prenatal testing for CF.

The majority of CF families may proceed as they did before prenatal diagnosis became available: they will either curtail reproduction (as many do) or take their chances. However, in the wider spectrum of abortion attitudes, the majority would abort for severe MR and many for a child bedridden for life. Optimism about CF was pervasive; families expected their child to reach age 40, hold a full-time job, and marry. With discovery of the gene for CF, optimism has probably increased.

At the other end of the spectrum, few would abort for adult-onset disorders starting at age 40 or 60, for genetic susceptibility to alcoholism, or for sex selection, areas now receiving much attention. Although overall responses suggest that most would not use abortion frivolously, responses to the obesity question aroused our concern. Apparently, there is a not insubstantial minority who would abort for cosmetic purposes if prenatal assessment were possible.

The trimester of pregnancy had less relationship to personal willingness to abort than might be expected. For most fetal conditions, including CF, only 5% fewer women would abort in the second trimester than in the first. This result suggests that newer methods of prenatal diagnosis, such as CVS, which make first-trimester abortions possible, may have little effect on deeply held attitudes.

TABLE 2—Stepwise Logistic Regression for Abortion for Cystic Fibrosis

Variable	Beta	P	Odds Ratio[a]
Siblings approve abortion for CF (3-point scale)	1.0319	.05	7.88
Spouse approves abortion for CF (3-point scale)	0.9396	.002	6.55
Infrequent attendance at religious services (4-point scale: once a year to weekly)	0.5791	.002	5.68
Spouse would not support carrying a CF fetus to term (3-point scale)	0.7713	.004	4.68
CF doctor approves abortion for CF (3-point scale)	0.3105	.08	1.86

[a] Odds that a respondent at one end of the variable scale will abort, when compared with a respondent at the other end.
Note: n = 222.

In genetic counseling, it would be appropriate to acknowledge the effects of extended family networks on patients' views and to encourage patients to discuss the relationship between personal and family values. The attitude of the physician caring for the affected child may influence parents' decisions. Few believed that their CF doctors would support abortion of a fetus with CF. Counselors should discuss these parental perceptions, which may reflect the clinically necessary optimism conveyed by pediatricians, rather than the doctors' actual views. Finally, the relationships between sociodemographic var-

TABLE 3—Stepwise Logistic Regression for Abortion for Severe Mental Retardation (n = 213)

Variable	Beta	*P*	Odds Ratio[a]
Siblings approve abortion for CF (3-point scale)	0.646	.0003	3.64
Respondent's mother approves abortion for CF (3-point scale)	0.5901	.002	3.26
Had previous therapeutic abortion	1.267	.04	3.55
Infrequent attendance at religious services (4-point scale: once a year to weekly)	0.2029	.097	1.83

[a] Odds that a respondent at one end of the variable scale will abort, when compared with a respondent at the other end.

iables and willingness to abort for fetal conditions reinforce concerns that differential use of selective abortion could make genetic disability a mark of social class and lead to further discrimination. ☐

Acknowledgments

This project was supported by the New England Regional Genetics Group, with funds from the Bureau of Maternal and Child Health Resources Development, DHHS. Timothy C. Heeren, Epidemiology and Biostatistics Section, Boston University School of Public Health, assisted with final revisions.

This article was presented at the American Society of Human Genetics, Cincinnati, Ohio, October 19, 1990.

References

1. Elkins TE, Stoval TG, Wilroy S, Dacus JV. Attitudes of mothers of children with Down syndrome concerning amniocentesis, abortion, and prenatal genetic counseling techniques. *Obstet Gynecol.* 1986;68:181–184.
2. Lubs ML, Falk RF. Response to prospective genetic counseling in severe X-linked disorders. *In:* Lubs HA, de la Cruz F, eds: *Genetic Counseling.* New York: Raven Press; 1977:269–289.
3. Evans DIK, Shaw A. Attitudes of hemophilia carriers to fetoscopy and amniocentesis. *Lancet.* 1979;2:1371.
4. Markova I, Forbes CD, Inwood M. The consumers' views of genetic counseling of hemophilia. *Am J Med Genet.* 1979; 17:741–752.
5. Francis RB, Kasper CK. Reproduction in hemophilia. *JAMA.* 1983; 250:3192–3195.
6. Hoyer LW, Carta CA, Golbus MS, Hobbins JC, Mahoney MJ. Prenatal diagnosis of classic hemophilia (hemophilia A) by immunoradiometric assays. *Blood.* 1985; 65:1312–1317.
7. Miller CH, Hilgartner MW, Aledort LM. Reproductive choices in hemophilic men and carriers. *Am J Med Genet.* 1987; 26:591–598.
8. Markel DS, Young AB, Penney JB. At-risk persons' attitudes toward presymptomatic and prenatal testing of Huntington disease in Michigan. *Am J Med Genet.* 1987; 26:295–305.
9. Meryash DL, Abuelo D. Counseling needs and attitudes toward prenatal diagnosis and abortion in fragile-X families. *Clin Genet.* 1988;33:349–355.
10. Beeson D, Golbus MS. Decision making: whether or not to have prenatal diagnosis and abortion for X-linked conditions. *Am J Med Genet.* 1985;20:107–114.
11. Golbus MS, Loughman WD, Epstein CJ, Halbasch G, Stephens JD, Hall BD. Prenatal diagnosis in 3,000 amniocenteses. *N Engl J Med.* 1979;300:157–163.
12. Benn PA, Hsu LYF, Carlson A, Tannenbaum HL. The centralized prenatal genetics screening program of New York City: III, the first 7,000 cases. *Am J Med Genet.* 1985;20:369–384.
13. Faden RR, Chwalow AJ, Quaid K, et al. Prenatal screening and pregnant women's attitudes toward the abortion of defective fetuses. *Am J Public Health.* 1987;77:288–290.
14. Boat TF, Welsh MJ, Beaudet AL. Cystic fibrosis. *In:* Scriver CF, Beaudet AL, Sly WS, Valle D, eds. *The Metabolic Basis of Inherited Disease,* 6th ed. Vol. II. New York: McGraw-Hill; 1989:2649–2680.
15. Brock DJH, Bedgood D, Barron L, Haywood C. Prospective prenatal diagnosis of cystic fibrosis. *Lancet.* 1985;1:1175–1178.
16. Farrall M, Law HY, Rodeck CH. First-trimester prenatal diagnosis of cystic fibrosis with linked DNA probes. *Lancet.* 1986; 1:1402–1404.
17. Evers-Kiebooms G, Denayer L, Van den Berghe H. A child with cystic fibrosis: II, subsequent family planning decisions, reproduction and use of prenatal diagnosis. *Clin Genet.* 1990;37:207–215.
18. Al-Jader LN, Goodchild MC, Ryley HC, Harper PS. Attitudes of parents of cystic fibrosis children towards neonatal screening and antenatal diagnosis. *Clin Genet.* 1990;38:460–465.
19. Boue A, Muller F, Nezelof C, et al. Prenatal diagnosis in 200 pregnancies with a 1-in-4 risk of cystic fibrosis. *Hum Genet.* 1986;74:288–297.
20. Elias S, Annas GJ. *Reproductive Genetics and the Law.* Chicago, Ill: Yearbook Publishers; 1989.
21. Evans MI, Fletcher JC, Drugan A, Bottoms SF, Platt LD. Ethics of selective termination: a survey of physicians of the American Society of Human Genetics (ASHG). *Am J Hum Genet.* 1988;43:A233.
22. Wertz DC, Fletcher JC. Ethical problems in prenatal diagnosis: a cross-cultural survey of medical geneticists in 18 nations. *Prenat Diagn.* 1989;9:145–157.
23. Wertz DC, Fletcher JC. Fatal knowledge? Prenatal diagnosis and sex selection. *Hastings Cent Rep.* 1989;19:21–27.
24. Kyle D, Cummins C, Evans S. Factors affecting the uptake of screening for neural tube defect. *Br J Obstet Gynaecol.* 1988; 95:560–564.
25. Luker K. *Abortion and the Politics of Motherhood.* Berkeley, Calif: University of California Press; 1983:158–215.
26. Evers-Kiebooms E. Decision making in Huntington's disease and cystic fibrosis. *Birth Defects.* 1987;23:115–149.
27. Swerts A. Impact of genetic counseling and prenatal diagnosis for Down syndrome and neural tube defects. *Birth Defects.* 1987;23: 61–83.
28. Cystic Fibrosis Foundation. *National Registry of Cystic Fibrosis Patients.* Rockville, Md; 1990.
29. Colten HR. Screening for cystic fibrosis: public policy and personal choices. *N Engl J Med.* 1990;322:328–329.
30. Kaback M, Zippin D, Boyd P, Cantor R. Attitudes toward prenatal diagnosis of cystic fibrosis among parents of affected children. *In:* Lawson D, ed. *Cystic Fibrosis: Horizons. Proceedings of the 9th International Cystic Fibrosis Congress, June 9–15, 1984.* Chichester, N.Y.: Wiley; 1984:15–28.
31. Denning CR, Gluckson MM. Psychosocial aspects of cystic fibrosis. *In:* Taussig LM, ed. *Cystic Fibrosis.* New York: Theeme-Stratton; 1984:461–492.

Infant Diet and Salmonellosis

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ABSTRACT

Background: The Island of Guam has experienced a high incidence of infant salmonellosis for a number of years (age-specific incidence rate of approximately 3700 cases per 100 000 infants in 1984). Interviews of case parents suggested that the use of high-iron infant formula was associated with this problem.

Methods: A case-control study was conducted to test this hypothesis. Information on feeding practices and a variety of medical and socioeconomic factors was collected for 78 laboratory-confirmed *Salmonella*-case infants and 167 control infants.

Results: Case infants were less likely to have been breast-fed (odds ratio [OR] = 9.15; 95% confidence interval [CI] = 2.71–30.9) and more likely to have been fed infant formula with an iron content of 10 mg/L or greater (OR=2.96; 95% CI = 1.24–7.08) than were control infants.

Conclusions: Although the precise means by which infants are most commonly exposed to *Salmonella* bacteria remain obscure, breast-feeding apparently protects against the development of physician-diagnosed clinical salmonellosis, while the feeding of high-iron infant formula has the opposite effect. (*Am J Public Health.* 1991;81:997–1000)

Introduction

The incidence of nontyphoid salmonellosis on the island of Guam increased from 78.6 cases per 100 000 population in 1975 to a peak of 218 cases per 100 000 in 1984. The incidence has declined to 110 per 100 000 in 1989, still more than 5 times the US rate for the same year.[1] Infants typically account for about 50% of *Salmonella* cases on Guam. Another unusual characteristic of the epidemic is the presence of *S. waycross,* a serotype that is rare elsewhere in the world but accounts for about 30% of all human *Salmonella* isolates on Guam. In 1984, only 3 of 35 862 cases of salmonellosis reported in the United States were due to *S. waycross;*[2] all three cases were residents of Guam who were referred to the state of Hawaii for medical treatment (A. P. Liang, personal communication, 1987).

Several investigations have failed to link infant cases of this epidemic to the consumption of foods with an animal origin (including milk), to secondary spread from infected family members, or to the ownership of pets or domestic animals.[3–5] Infant salmonellosis cases occur with approximately equal frequency in both civilian and military communities as well as in families of both higher and lower economic status.[3] Investigations of infant day care and formula preparation practices have not provided any clues to the origin of infant salmonellosis cases.[4]

During the course of an earlier unpublished study of this problem, we were impressed by an apparent association between infant salmonellosis cases and a history of being fed iron-supplemented brands of infant formula. Samples of these products, obtained both from case households and unopened from grocery shelves, were invariably sterile, however.

In 1984 an article in the veterinary literature reported that weanling pigs administered therapeutic amounts of iron orally developed more severe *Escherichia coli* diarrhea than pigs to which the same amounts of iron were administered by intramuscular injection; the authors theorized that increased amounts of available iron in the intestinal tract promoted the multiplication of otherwise harmless numbers of potentially pathogenic bacteria, thus rendering them better able to cause overt disease.[9]

Since iron-enriched formula is commonly fed to infants, we initiated a study to determine if the iron content of formula could be contributing to the problem of infant salmonellosis on Guam.

Methods

A case-control study was carried out using as cases, infants born on Guam who developed laboratory-confirmed salmonellosis before their first birthday. Cases were identified prospectively as laboratory reports were received from the Guam public health reference laboratory. (All medical laboratories on Guam, including

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This paper was submitted to the journal May 17, 1990, and accepted with revisions February 19, 1991.

TABLE 1—Crude odds ratios (OR) for Iron Content of Formula Fed at Onset of Illness in Non-Breast-Fed Cases of Infant Salmonellosis and Non-Breast-Fed Age-Matched Controls

Formula	Cases	Controls	OR*	95% CI
Low-iron	13 (17.3%)	37 (30.0%)	1.00	
High-iron	62 (82.7%)	88 (70.0%)	2.96	1.24–7.08

*OR and 95% CI estimated retaining age-matching.

military facilities, routinely submit isolates of *Salmonella, Shigella,* and *Vibrio* to this laboratory for confirmation and serotyping.)

Controls were identified by selecting the next two births listed in the Guam birth registry following the case-birth registration, thereby matching for age and season of birth. If an infant selected as a control was deceased, or a salmonellosis case, or had previously been selected as a control, the next registered birth was selected. If a selected infant had been treated by a physician for diarrhea or if efforts to locate parents were not successful, the next birth certificate was selected, and the process was repeated until two controls had been enrolled in the study.

Parents of case infants were asked what milk their infant was being fed when the diarrhea developed, and parents of control infants were asked what milk their infant was being fed at the same age. Formula brands were assigned to one of two categories: low iron (less than 10 mg/L) or high iron (10 mg/L or more). Upon completion of each interview, data on the age, ethnicity, education, and marital status of the parents, infant birthweight and APGAR score, complications of pregnancy or delivery, month of pregnancy during which prenatal care began, number of prenatal physician visits, and number of previous births to the mother were excerpted from birth certificates.

Names of all study infants, without identification as to whether they were cases or controls, were submitted to the Special Supplemental Food Program for Women, Infants and Children (WIC) staff to determine if they had been enrolled in that program.

Statistical Methods

Cross-tabulations and simple statistical tests, including χ^2 tests, were performed using the computer package PC-SAS.[10] Odds ratios (OR) and their 95% confidence limits (CI) were estimated by conditional logistic regression analysis, retaining the matching of controls to cases as suggested by Breslow and Day,[11] using the computer package EGRET.[12]

Results

Of 96 cases of salmonellosis reported in children less than 1 year of age during this study, 18 (18.8%) could not be traced. In selecting controls, birth certificates of 389 infants were examined. Of these, 131 (34%) were excluded from the study for various reasons, 112 being untraceable. Of the remaining 258 controls, 167 had no history of treatment by a physician for diarrhea and were enrolled in the study.

Cases were less likely to be receiving breast milk at the time they developed salmonellosis than their age-matched controls (3.9% vs 25.2%), suggesting that failure to breast-feed infants on Guam is associated with an approximately ninefold increase in the risk of symptomatic salmonellosis infection (OR = 9.15, CI = 2.71–30.9). This association remained after taking into account potentially confounding variables.

To compare infants with regard to the iron content of infant formula received, those infants receiving breast milk were excluded, leaving a total of 75 cases and 125 controls. Cases were more likely than controls to be receiving infant formula with a high iron content (OR = 2.96, CI = 1.24–7.08; Table 1). Ninety-six percent of cases enrolled in the WIC program received a high-iron formula compared with 75% of cases not enrolled. Among controls, 80% of infants in the WIC program received a high-iron formula compared with 67% of those not in the program. No association between education of parents or marital status of mother and type of infant formula used was observed.

Each of the variables included in Table 2 was then considered individually as a potential confounder of the association between type of infant formula and risk for salmonellosis using the technique of conditional logistic regression. Controlling WIC enrollment led to the greatest reduction in the estimate of the odds ratio (OR = 2.54, CI = 1.02–6.28), while controlling marital status led to the greatest increase (OR = 3.85, CI = 1.51–9.84). A conditional logistic regression model including the variables listed in Table 2 was then fitted. Problems of collinearity prevented the simultaneous inclusion in the model of terms for father's ethnic group and age. All other variables were included. When father's ethnic group was omitted from the model, the estimated OR of the association between high-iron infant formulas and salmonellosis was 4.74 (CI = 1.28–17.57). Excluding father's age from the model produced an OR estimate of 4.04 (CI = 1.09–14.94).

Discussion

It has been suspected for some time that infants born on Guam may be infected with *Salmonella* bacteria by some means other than the more commonly recognized modes of transmission. The discovery that both indoor and outdoor environments on Guam were heavily contaminated with *Salmonella* bacteria[13,14] and that the incidence of infant salmonellosis in particular was linked to seasonal changes in environmental conditions[15] seemed to support the hypothesis that infant salmonellosis might frequently be a consequence of contamination of the infant's environment rather than the result of consuming contaminated food products or contact with infected family members.[16] It was also suspected that the immunologic naivete of newborn infants was a key factor in facilitating infection by the relatively small number of *Salmonella* bacteria that were likely to be encountered in an apparently clean home environment and that were harmless to other family members. The results of this study suggest that absence of breast-feeding and high levels of iron in some brands of infant formula may also be factors that contribute to an increased risk for infants' developing clinical salmonellosis on Guam.

Reported complications associated with pregnancy or delivery was the only other variable to show a substantially elevated OR in our univariate analysis (Table 2). This trend remained in both multivariate models (OR = 2.79, CI = 0.76–10.26, father's ethnic group excluded, and OR = 3.68, CI = 1.08–12.55, father's age excluded). Complications reported in more than one case included wrapping of umbilical cord around neck of infant (14), meconium staining of infant (8), Caesarean delivery (6), and premature rupture of membranes (2). These infants may have been at increased risk of being identified as salmonellosis cases because of decreased resistance to infection or because they received more intensive medical attention following delivery. While we did not measure either disease-specific or general infant immunity levels, our analysis did use the number of antenatal physician visits as a proxy measure of the tendency of parents to utilize medical services.

Other possible biases in this study include loss of 18 (18.9%) cases and 11 (28.8%) controls. Factors that contributed to the high rate of unlocateable cases and controls included the mobility of younger families, the concurrent use of as many as three different house-numbering schemes in some areas, and the frequent absence of addresses or telephone numbers in patient records. The fact that case address information obtained from health care providers was more current than control address information listed on birth certificates may have affected the success rates for locating individuals from these two groups.

Of initially selected controls, 91 were excluded from the analysis because they had a history of physician treatment for diarrhea and might therefore have been unidentified cases of salmonellosis. Trial inclusion of these children in the analysis led to some reduction in the final estimate of the OR of the association between type of infant formula and risk of salmonellosis, as might be expected if misclassification of disease status had occurred (OR = 2.34, CI = 1.22–9.16), but would not materially alter our interpretation of the results.

A biased estimate of the OR might also have arisen through failure to control other confounding variables. While socioeconomic status has not been controlled directly in the analysis, maternal and paternal education, both indicators of socioeconomic status, have been controlled. Enrollment in the WIC program, which is restricted to low-income families, has also been controlled. Cost differential between low- and high-iron formulas is not a factor, as both formulations cost the same.

Weaning practices could also be a confounding factor, but since 60% of children in the study were age 3 months or less, it seems unlikely that this could have played a substantial role in the observed associations. Formula preparation practice was not examined because an earlier investigation suggested that this was not a risk factor for infant salmonellosis on Guam.[4]

TABLE 2—Crude odds ratio (OR) for Various Physical, Demographic, and Socioeconomic Variables: Infant Salmonellosis Cases vs Age-Matched Controls

	Cases		Controls			
Variable	No.	Percentage	No.	Percentage	OR	95% CI
Sex						
Males	48	62	97	58	1.00	
Females	30	38	70	42	0.89	0.51–1.57
Birth order						
First	30	38	60	36	1.00	
Later	48	62	106	64	0.90	0.53–1.56
Birth weight (g)						
2500 or more	72	92	157	94	1.00	
<2500	6	8	10	6	1.25	0.43–3.60
Mother's status						
Married	61	78	114	68	1.00	
Unmarried	17	22	53	32	0.62	0.33–1.15
Ethnic group of mother						
Pacific Islander	44	56	90	54	1.00	
Asian	18	23	48	29	0.86	0.44–1.67
Other	16	21	29	17	1.22	0.52–2.84
Ethnic group of father						
Pacific Islander	36	52	56	43	1.00	
Asian	17	25	37	29	0.79	0.38–1.65
Other	16	23	36	28	0.67	0.27–1.62
Complications of pregnancy or delivery						
No	44	56	116	69	1.00	
Yes	34	44	51	31	2.10	1.07–4.15
Hospital of birth						
Civilian	59	76	122	73	1.00	
Military	19	24	45	27	0.87	0.30–2.50
Mother's age (years)						
<20	8	10	23	12	1.00	
20–29	49	63	106	64	1.24	0.52–2.95
≥30	21	27	36	24	1.68	0.65–4.37
Father's age (years)						
<20	4	6	5	4	1.00	
20–29	33	49	69	53	0.63	0.16–2.41
≥30	31	46	55	43	0.73	0.18–2.87
Mother's education						
Secondary	47	60	112	67	1.00	
College	31	40	54	33	1.43	0.81–2.52
Father's education						
Secondary	38	60	76	60	1.00	
College	25	40	50	40	1.07	0.57–2.03
Number of prenatal visits						
1–8	30	38	63	38	1.00	
9–11	24	31	56	34	0.95	0.48–1.89
≥12	24	31	46	28	1.12	0.51–2.44
APGAR score at 1 min						
1–8	53	68	113	68	1.00	
9–10	25	32	52	32	1.03	0.56–1.91
APGAR score at 5 min						
1–8	11	14	21	13	1.00	
9–10	67	86	144	87	1.00	0.41–2.45
WIC client						
No	51	65	130	78	1.00	
Yes	27	35	37	22	1.74	0.94–3.21

Note: OR and 95% CI estimated retaining age-matching.

It has previously been reported that breast-feeding and available dietary or serum iron can influence the course of infections.[17–22] Since mother's milk contains only about 1.5 mg of iron per liter and normal full-term infants exclusively breast-fed for 4 to 6 months by mothers in normal health are unlikely to develop iron deficiency,[23] it may be that maintaining a relatively low concentration of iron in the neonatal gastrointestinal tract is one of nature's strategies for protecting infants from bacterial infections during the critical first few months of life. Commercial iron-supplemented infant formula, on the other hand, contains from 12 to 19 mg of iron per liter and would provide the intestinal flora of infants fed these products with from 8 to 12 times more iron than is available to breast-fed infants.

Although one of the objectives of the Guam WIC program is to encourage breast-feeding, this study found that breast-feeding was less common among WIC program participants than among nonparticipants (19% vs 28% among controls). The enticement of offering free infant formula to new mothers may be more effective in determining actual nursing practice than present educational efforts designed to foster knowledge and attitudes favorable to breast-feeding. Current regulations of the WIC program stipulate that infant formula containing less than 10 mg of iron per liter may be provided only when a physician determines that the recipient infant has an existing medical condition that contraindicates the use of iron-supplemented formula. This study suggests that such a requirement may contribute to the problem of infant salmonellosis on Guam.

For the 10-year period 1981 to 1990, the average annual age-specific incidence rate for infant salmonellosis on Guam peaked at age 2 months (approximately 4580 cases per 100 000) but fell by more than 50% by age 6 months (approximately 2140 cases per 100 000). This result suggests that it would be advisable to breast-feed Guam newborns for at least 6 months and to delay feeding iron-supplemented infant formula for the same period when breast-feeding is not possible.

While we have only studied the relationship between salmonellosis and infant diet, it is possible that other bacterial diseases are similarily affected. It has already been demonstrated using an animal model that oral iron supplementation can cause an exacerbation of diarrhea due to *E. coli* infection,[9] and both pathogenic strains of *E. coli* and *Campylobacter* (a bacteria similar epidemiologically to *Salmonella*) are known to be important causes of childhood diarrhea in many countries.[24–26]

Although a relatively high nonresponse rate among both cases and controls and the other potential sources of bias outlined in this report suggest that a cautious interpretation of our conclusions is warranted, we believe that the data presented here, together with a biologically plausible mechanism previously reported, warrant further investigation. Certainly any inexpensive and readily implemented strategies that have the potential to significantly affect the important worldwide problem of infant diarrheal disease merit serious consideration. □

Acknowledgment

This work was supported by grant WP 82005 from the Diarrhoeal Diseases Control Program of the World Health Organization, Geneva.

References

1. Centers for Disease Control. Annual summary of notifiable diseases, 1989. *MMWR.* 1989:38:5–4.
2. Centers for Disease Control. *Salmonella Surveillance Report 1984.* Atlanta, Ga; 1984.
3. Haddock RL, Delon PJ. Salmonella surveillance in Guam. *Southeast Asian J Trop Med Public Health.* 1980;11:48–54.
4. Haddock RL, Malilay J. A search for infant salmonellosis risk factors on Guam. *Southeast Asian J Trop Med Public Health.* 1986;17:38–42.
5. Diarrhoeal Diseases Control Program, World Health Organization. Salmonellosis in Guam. *WHO Weekly Epidemiol Rev.* 1983;59:119–122.
6. Benenson AS, ed. *Control of Communicable Diseases in Man.* 14th ed. Washington, DC: American Public Health Association; 1985.
7. Feldman RA. Salmonellosis. *In:* Last JM, ed. *Public Health and Preventive Medicine.* New York: Appleton-Century-Crofts; 1980:427–431.
8. Krugman S, Ward R, Katz SL. *Infectious Diseases of Children.* Saint Louis: Mosby; 1977:300.
9. Kadis S, Udeze FA, Palanco J, Dreesen DW. Relationship of iron administration to susceptibility of newborn pigs to enterotoxic colibacillosis. *Am J Vet Res.* 1984;45:255–259.
10. SAS Institute, Inc. *SAS/SAT Guide for Personal Computers, Version 6.* Cary, NC; 1985.
11. Breslow NE, Day N. *Statistical Methods in Cancer Research. 1: The Design and Analysis of Case-Control Studies.* Lyon: International Agency for Research on Cancer; 1980. Publication No. 32.
12. Statistics and Epidemiology Research Corporation. EGRET. Seattle, Wash; 1989.
13. Haddock RL, Nocon FA. *Salmonella* contamination of soil in children's play areas on Guam. *J Environ Health.* 1986;49:158–160.
14. Haddock RL. Salmonella in vacuum cleaners [letter]. *Lancet.* 1986;11:637.
15. Haddock RL, Malilay J. The possible role of rainfall in spreading *Salmonella* on Guam. *J Diarrhoeal Dis Res.* 1986;4:229–232.
16. Haddock RL. Three modes of *Salmonella* transmission. In: Muangman D, ed. *Proceedings of the Second International Symposium on Public Health in Asia and the Pacific Basin.* Bangkok: Mahidol University; 1986:191–195.
17. Kovar MG, Serdula MK, Marks JS, Fraser DW. Review of the epidemiologic evidence for an association between infant feeding and infant health. *Pediatrics.* 1984;74:615–638.
18. Feachem RG, Koblinsky MA. Interventions for the control of diarrhoeal disease among young children: promotion of breast-feeding. *Bull World Health Organ.* 1984;62:271–291.
19. Bullen JJ, Rogers HJ, Leigh L. Iron-binding proteins in milk and resistance to *Escherichia coli* infection in infants. *Br Med J.* 1972;1:6975.
20. Bullen JJ, Rogers HJ, Griffiths E. Bacterial iron metabolism in infection and immunity. In: Neilands JB, ed. *Microbial Iron Metabolism.* San Diego, Calif.: Academic Press; 1974;517–551.
21. Kluge MJ, Rothenburg BA. Fever and reduced iron: their interactions as a host defense to bacterial infection. *Science.* 1979;203:374–376.
22. France GL, Marmer DJ, Steele RW. Breast-feeding and *Salmonella* infection. *Am J Dis Child.* 1980;134:147–152.
23. Miller A, Chopra JG. Problems with human milk and infant formulas: report of the task force on the assessment of the scientific evidence relating to infant-feeding practices and infant health. *Pediatrics.* 1984;74:639–647.
24. Diarrhoeal Disease Control Program, World Health Organization. *Studies on the Etiology of Acute Diarrhoea.* Geneva; 1986. Publication CDD/28.2.86.
25. Tauxe RV, Pegues DA, Hargrett-Bean N. Campylobacter infections: the emerging national pattern. *Am J Public Health.* 1987;77:1219–1220.
26. Black RE, Romana GL, Brown KH, et al. Incidence and etiology of infantile diarrhea and major routes of transmission in Huascar, Peru. *Am J Epidemiol.* 1989;129:785–799.

Controlled Trial of *Giardia lamblia*: Control Strategies in Day Care Centers

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ABSTRACT

Background: Strategies for control of *Giardia lamblia* in day care differ in numbers of children treated and in costs to parents and day care operators. The effectiveness of these strategies has not been systematically evaluated.

Methods: We conducted a prospective randomized controlled trial comparing three strategies for control of *Giardia* in infant-toddler day care centers: Group 1, exclusion and treatment of symptomatic and asymptomatic infected children; Group 2, exclusion and treatment of symptomatic infection only; Group 3, exclusion and treatment of symptomatic infection, treatment of asymptomatic infection in the center. The study included 31 day care centers with 4180 child-months of observation. *Giardia* prevalence was determined before intervention and 1, 2, 4, and 6 months later; new infants and toddlers were tested on admission.

Results: Initial *Giardia* prevalences were 18% to 22% in the three groups. *Giardia* was identified in 10.5% of 676 new infants and toddlers entering study day care centers during the 6-month follow-up. *Giardia* prevelances by intervention group were 8%, 12%, and 7% at 1 month, and 7%, 8%, and 8% at 6 months.

Conclusions: The stricter intervention resulted in greater cost in terms of child day care and parents' work days lost, but did not result in significantly better control of *Giardia* infections in this day care environment. (*Am J Public Health.* 1991;81:1001–1006)

Introduction

Giardia lamblia infection and diarrhea have been increasingly identified in association with child day care centers, especially those caring for infants and toddlers: day care center outbreaks have been reported from Atlanta, Tucson, Houston, and Phoenix, with 17–54 percent of children in these outbreaks found to be infected.[1–4] A prospective study in Phoenix, Arizona day care centers found *Giardia* to be the most frequently identified enteric pathogen in diarrhea outbreaks and in sporadic diarrhea among infants and toddlers.[4]

Asymptomatic children in day care centers where *Giardia* diarrhea is identified are also frequently infected.[1,2,5] These asymptomatic infections are of theoretical concern in developing disease control strategies because asymptomatic carriers often excrete the infective cyst form of the parasite.[5,6] However, the actual relation of such asymptomatic excretion to *Giardia* transmission in day care centers has not been documented.

For almost 10 years, the Maricopa County (Greater Phoenix) Department of Health Services had utilized a very strict intervention strategy to control giardiasis in centers where the parasite was identified by passive laboratory-based surveillance and case investigation:

- If the case is a day care center child or worker, all children and staff were tested for *Giardia*.
- All symptomatic and asymptomatic *Giardia*-infected children and adults were excluded from the center, with recommendation for treatment.
- Excluded children or staff were readmitted to the center when treatment was completed and two *Giardia*-negative stool examinations had been documented.
- The center was given advice regarding hygienic practices to reduce transmission of enteric microorganisms.

Although this strategy had substantial impact on families and centers involved, its effectiveness in *Giardia* control had not been evaluated. We therefore conducted a controlled study of the effectiveness of this strategy in comparison with other strategies for control of *Giardia lamblia* infection among infants and toddlers in Phoenix day care centers.

Methods

General

This was a prospective randomized (unblinded) controlled trial. All centers having a case of *Giardia lamblia* infection identified in an infant (age 0–11 months) or toddler (age 12–35 months) during the period October 1986–September 1987 were considered for inclusion in the study. Permission of the director and parents was required for participation. On acceptance of participation, each center was assigned to one of the three control strategies, using prenumbered sealed envelopes containing the group assignment derived from a table of random numbers according to a block randomization scheme. If a center de-

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clined participation, *Giardia* control was managed according to the existing policy and excluded from study and analysis. This study was approved by the Committee on Human Subjects of the Maricopa County Department of Health Services.

Control Strategies (Intervention Groups)

Group 1. Exclusion and recommendation of treatment for symptomatic and asymptomatic infections; readmission after completion of treatment, and two *Giardia*-negative stool examinations by the health department (existing strategy).

Group 2. Exclusion and recommendation of treatment for symptomatic infections only; readmission when asymptomatic, with continued treatment and follow-up testing in the center. No exclusion or treatment of asymptomatic infections.

Group 3. Exclusion and recommendation of treatment for symptomatic infections; readmission when asymptomatic, with continued treatment and follow-up testing in the center. Treatment and follow-up testing of asymptomatic infections in the center, without exclusion.

All groups received the same standard orientation regarding hygiene practices to reduce spread of *Giardia*.

Outcome Variables

Effectiveness of the intervention strategies was assessed by comparing prevalences of *Giardia* among infants and toddlers at 1, 2, 4, and 6 months after intervention, and occurrence of *Giardia*-positive diarrhea among infants and toddlers during intervals between follow-up prevalence test rounds.

Data Collection

Information on each participating center included total enrollment, number of infants (if present) and toddlers, type of organization, operating hours, child group mixing, staff supervising more than one child group, and location within Phoenix (zip code sector). Standardized observations regarding sanitary and child care practices were made by study nurses during the six-month follow-up period in each center. Information on each participating child was obtained at the time of center entry into the study for children enrolled at that time (original enrollees), or on entry to the center for children enrolling during the six-month follow-up period (new enrollees). These data included age, sex, ethnic group, attendance status (regular, drop-in), duration of enrollment (original enrollees) or previous day care (new enrollees), other concurrent day care, parent occupation, household size, presence of siblings by age group, home location (zip code sector), pets, exposure to untreated water (by type), and travel history. Information subject to short-term change was re-ascertained by interview at the time of follow-up test rounds.

Giardia lamblia *Prevalence Testing*

Consistent with existing procedures, *Giardia* infection was determined by examination of a single stool specimen collected by the center or parents and tested in the health department laboratory. Prevalence testing at each specified interval was conducted during a period of two weeks. A minimum testing compliance standard (at least 75 percent of enrolled infants and toddlers) was maintained for all test rounds and, when necessary, the test period was extended until this testing level was obtained. In almost all follow-up prevalence testing rounds this level was exceeded, and the mean proportion of enrolled children tested in follow-up test rounds was 83 percent.

New entry children were initially tested within two weeks of admission. In subsequent test rounds, children enrolling after intervention were retested along with original enrollees. Results of initial *Giardia* tests of original enrollees were known to study nurses, center staff, and parents. Study and center personnel and parents were blinded to results of new enrollee testing and of follow-up test rounds for all children.

Surveillance for Diarrhea

Nurse field workers made daily telephone calls to each study center to identify cases of diarrhea (stools looser and more frequent than normal for that child) among infants and toddlers, and to obtain attendance figures. Visits to each center were made at least weekly to deliver specimen collection materials, reinforce study methods, and verify enrollment. When diarrhea occurred in a study child between follow-up test rounds, a stool specimen was collected for *Giardia* testing. Test results of children with diarrhea were known to study personnel; if *Giardia*-positive, children with diarrhea were managed according to the control strategy for their center.

Parasitologic Methods

Fresh stool specimens were immediately placed in two vials, one containing polyvinyl alcohol (PVA) and the other 10 percent formalin, by the parent or center worker. All specimens were examined by ethyl-acetate-formalin concentration of the formalin-preserved stool, followed by staining with D'Antoni's modified iodine and direct microscopic examination at 100× and 400× magnification. Specimens from children with diarrhea were also examined for *Giardia* trophozoites using trichrome staining of the PVA-preserved stools. For quality control, a subset of specimens was randomly selected and also tested using an indirect immunofluorescent assay for the detection of *Giardia* cysts in feces (Merifluor-*Giardia*, Meridian Diagnostics, Cincinnati, Ohio).

Potential Confounding Variables

Variables which might affect the rate of *Giardia* infection among the children and centers in the different intervention groups were identified from review of pertinent literature. We performed two analyses to assess the association of these variables with *Giardia* infection among Phoenix infants and toddlers, and to examine additional local variables (such as geographic sector of the city or exposure to untreated water used for irrigation of some yards and public parks) which might be associated with infection. The first analysis was a case-control study comparing the 51 new enrollee children without previous day care attendance who were found to be *Giardia*-infected on admission to study centers (cases) with the 482 new enrollee children without previous day care who were not infected on admission (controls). A second analysis compared characteristics of 52 children age 0–35 months who had never attended day care and who were identified as *Giardia*-infected during the study period by health department surveillance, with 52 age-matched controls randomly selected from county birth records who also had never attended day care and who were confirmed as not infected by stool testing. The prevalences of confounding variables identified from the literature or by one of these analyses were then compared in the three intervention groups, to identify those which would need to be considered in analysis of the effect of intervention strategy on *Giardia* prevalence.

Association of center characteristics with prevalence of *Giardia* was examined by comparing the 14 study centers having initial *Giardia* prevalences above the mean for all study centers, with the 17 study centers having lower initial *Giardia* prevalences.

Statistical Analysis

Prevalences of *Giardia* among infants and toddlers in the centers in each of the three intervention groups at each of the specified test points were compared using ANOVA. Comparison of rates and proportions was performed using chi-square, Fisher's exact, and Wilcoxon-Mann-Whitney tests. To examine the effect of intervention group on *Giardia* prevalence, accounting for the effects of confounding variables, a nested ANOVA model was used.

In all of these analyses, the null hypothesis (that no significant association existed between an independent variable and the outcome under study) was rejected if the probability of chance association was <0.05.

Estimated Costs of Control Strategies

Our study was not designed to provide a detailed cost-effectiveness analysis of the various *Giardia* control strategies. However, we did attempt to estimate the costs of these strategies to the families actually participating in the study. Projections regarding the number of children requiring medical treatment, costs of these treatments, and number of day care and parent work days lost were developed for asymptomatic and symptomatic children under the different intervention strategies. These projections were then applied to the actual number of such children in each group at the time of intervention.

The projections assumed exclusion and treatment of all *Giardia*-positive children in accordance with the protocol for their treatment group. In Group 1, duration of exclusion of all *Giardia*-positive children was projected as two weeks or 10 day care days (10 days treatment, four days follow-up testing after treatment). In Groups 2 and 3, duration of exclusion of symptomatic *Giardia*-positive children was projected as one week (five day care days) until end of symptoms and readmission. To develop projections of treatment costs and parent work days lost we conducted a survey which included 38 percent of parents of study children who were *Giardia*-positive at the time of initial intervention.

Results

Centers Entering Study

During the one-year enrollment period, 35 centers with an index case of *Giardia lamblia* infection in the infant-toddler age group were identified. Of the 32 centers having additional cases of infant-toddler *Giardia* infection, all accepted participation; one center was dropped after intervention for failure to comply with the study protocol and its data were excluded from analysis. The remaining 31 centers completed six months of follow-up for a total 4,180 infant-toddler child-months of observation. Characteristics of these 31 centers are shown in Table 1, by intervention group.

TABLE 1—Characteristics of Study Day Care Centers, by Intervention Group

Characteristics	Group 1 N = 10 (%)	Group 2 N = 11 (%)	Group 3 N = 10 (%)
For-profit organization	9 (90)	9 (82)	10 (100)
Operate 50–65 hours/week	8 (80)	9 (92)	8 (80)
Accept public agency payment	10 (100)	10 (91)	8 (80)
Accept infants [age < 1 year]	6 (60)	9 (82)	4 (40)
Mean number of infant-toddlers at initial testing	26 (9)*	31 (10)*	30 (11)*
Drop-ins (% of infant-toddlers)	(1)	(<1)	(1)
Mean turnover index [# new infant-toddler enrollees during study + # original infant-toddler enrollees]	0.84 (0.10)*	0.82 (0.14)*	0.70 (0.10)*
Proportion of *Giardia*-positive children symptomatic at intervention	17%	39%	22%

*standard error.

TABLE 2—Prevalences of *Giardia Lamblia* on Initial and Post-intervention Testing among Infants and Toddlers in Study Day Care Centers, by Intervention Group and Enrollment Category

Intervention Group	Enrollment Category	Initial	1 month	2 months	4 months	6 months
1	Already enrolled*	20% (258)	7% (165)	7% (181)	8% (183)	6% (175)
	New**	—	10% (58)	11% (54)	9% (74)	9% (34)
	Total	20% (258)	8% (223)	8% (235)	8% (257)	7% (209)
2	Already enrolled*	18% (308)	12% (193)	14% (211)	12% (217)	6% (207)
	New**	—	12% (32)	6% (67)	8% (93)	15% (60)
	Total	18% (308)	12% (225)	12% (278)	10% (310)	8% (267)
3	Already enrolled*	22% (299)	6% (195)	10% (190)	8% (191)	7% (209)
	New**	—	12% (34)	13% (60)	10% (78)	14% (36)
	Total	22% (299)	7% (229)	11% (250)	8% (269)	8% (245)

*Already enrolled = Children enrolled at initial testing, or enrolled throughout the interval ending in each test round.
**New = Children enrolling during the interval ending in the test round.

Immediate Observations after Intervention

The overall prevalences of *Giardia* at the time of intervention did not differ significantly among the infants and toddlers in each of the intervention groups (20 percent in Group 1, 18 percent in Group 2, and 22 percent in Group 3). The ranges of *Giardia* prevalences in the individual centers at intervention were 6–32 percent in Group 1, 8–30 percent in Group 2, and 5–47 percent in Group 3.

Table 2 presents overall *Giardia* prevalences by intervention group among children already enrolled (prior to the intervention for initial testing and during the entire interval between tests for each subsequent test round) and for children newly enrolled during each of the intervals between test rounds. Several differences between intervention groups were noted in the first month after intervention. In Groups 1 and 3 there were significant reductions in overall *Giardia* prevalence at one month, to 8 percent and 7 percent, respectively. Although the overall prevalence in Group 2 also declined to 12 percent, the difference was less, consistent with the fact that this intervention did not treat asymptomatic *Giardia*-positive children.

In addition, all groups experienced drops in enrollment after initial identification of *Giardia* and intervention imple-

TABLE 3—Contribution of Original and New Enrollees to the Total Infant Toddler Study Population and to *Giardia Lamblia* Infection at Conclusion of Six Months' Follow-up, by Initial *Giardia* Test Result

Enrollment and Test Result Category	Proportion Six Months Post-Intervention: Total Infants and Toddlers (n = 721) %	Total *Giardia*-positive Infants and Toddlers (n = 56) %
Original Enrollees		
Initially *Giardia*-positive	12	18
Initially *Giardia*-negative	43	18
New Enrollees		
Initially *Giardia*-positive	4	39
Initially *Giardia*-negative	41	25

TABLE 4—Episodes of Diarrhea and *Giardia*-positive Diarrhea Occurring in Study Infants and Toddlers between Follow-up Test Rounds, by Intervention Group

Intervention Group	Child-months between Test Rounds	Total Number of Diarrhea Episodes	Number of *Giardia*-Positive Episodes (%)
1	832	60	6 (10)
2	1,048	183	29 (16)
3	907	81	16 (20)

mentation. There were significant differences among intervention groups in the proportion of initially *Giardia*-positive children who left study day care centers in the first month after intervention: only 7 percent of *Giardia*-positive children dropped out of Group 3, compared to 25 percent in Group 1 and 20 percent in Group 2. The drop-out rates of *Giardia*-negative children in the first month after intervention in the three groups were 15, 14, and 16 percents, respectively.

Finally, compliance with post-treatment testing was significantly reduced in the groups where such testing was not required prior to re-admission: only 71 percent of children treated in Groups 2 and 3 submitted one or more post-treatment clearance specimens. Compliance with treatment may also have been different: of treated children submitting post-treatment specimens, 23 percent in Groups 2 and 3 remained *Giardia*-positive, compared to 4 percent in Group 1.

Six-Month Follow-Up

Prevalence of Giardia lamblia. As shown in Table 2 Groups 1 and 3 had similar overall prevalences of *Giardia* at each of the follow-up test points. Although *Giardia* prevalences in Group 2 were somewhat higher than those in the other two groups at the 1, 2, and 4 month follow-ups, by the six-month follow-up the overall *Giardia* prevalences in the three groups were virtually identical (7, 8, and 8 percents, respectively).

Contribution of new versus original enrollees and initial test status to outcome. Table 3 shows the contribution of initially *Giardia*-infected and uninfected original and new enrollee children to the total study population and to *Giardia* infection at the end of follow-up. Six months after intervention, original enrollees accounted for 55 percent of infants and toddlers and for 36 percent of *Giardia* infection. Children who had been *Giardia*-infected at the time of intervention were over three times more likely to be infected six months later than children originally not infected. The importance of originally *Giardia*-positive children in total infection after six months was actually greatest in Group 1, despite these children having been subject to the strictest intervention and post-treatment testing.

Initial testing of all new infant-toddler enrollees found 10.5 percent to be *Giardia*-positive upon entering study centers; this proportion did not differ significantly among intervention groups (10 percent in Groups 1 and 2, 12 percent in Group 3). At six months after intervention, 57–75 percent of *Giardia*-positive infants and toddlers in each group were children who had enrolled after the intervention. New enrollees who were *Giardia*-positive on admission comprised only 4 percent of the total infant-toddler population at six months post-intervention, but accounted for 39 percent of all *Giardia*-infected children.

Giardia-associated diarrhea between test rounds. Table 4 shows the number of diarrhea episodes identified between test periods in each intervention group, and the proportion of these episodes which were *Giardia*-positive. The overall rates of diarrhea and of *Giardia*-positive diarrhea were significantly higher in Group 2 than in Groups 1 and 3. However, the proportions of diarrhea episodes which were *Giardia*-positive were not significantly different among the three groups. Increased testing in response to a greater number of episodes of diarrhea might have resulted in detection of more "*Giardia*-positive" diarrhea in Group 2. However, the testing and treatment of symptomatic *Giardia*-positive children represents an additional control measure which would have had more effect in Group 2 than in Group 1 or 3.

Potential confounding variables. Five characteristics other than day care attendance were found to be significantly associated with infant-toddler *Giardia* infection in new enrollees without previous day care who entered study day care centers, or in the case-control study of *Giardia*-infected 0–35 month old children not attending day care. Comparison of the prevalences of these characteristics among study children in the three intervention groups found very low prevalences of two (Native American ethnic group, 2–5 percent; well water exposure, 1–2 percent). Two other characteristics were more common, but were almost equally prevalent in all groups (household size ≥ 5 persons, 25–29 percent; presence of a 3–5 year old sibling, 28–32 percent). Only one factor, the study child belonging to the toddler (12–35 month) age group (versus the infant age group), was significantly associated with *Giardia* infection and differed significantly in its distribution between the three intervention groups (78 percent in Group 1, 65 percent in Group 2, and 82 percent in Group 3, $p < 0.01$.). The lower proportion of toddlers in Group 2 would be expected to lower the observed prevalences of *Giardia*, while the higher proportion of toddlers in Group 3 would be expected to raise the observed prevalences of *Giardia* in that group.

Among all new enrollees, previous day care center (but not day care home) attendance was significantly associated with *Giardia* infection, but only 8 percent of new enrollees had previously attended a day care center.

No day care center characteristic was significantly associated with higher prevalence of *Giardia* on initial testing, although this analysis was limited by the small total number of centers.

Multivariate analysis. To examine the effects of the *Giardia* control strategies taking into account the differences in age distribution among the day care centers in the three groups, we employed a series of nested ANOVA models which included *Giardia* prevalence (at one of the specified test points for each model), intervention group, proportion of infants and toddlers tested at that point who were in the toddler age group, and individual day care center (nested within intervention group). In these models, the differences in proportion of toddlers were significantly associated with differences in *Giardia* prevalence at the time of initial testing and at 1, 2, and 4 months after intervention. Intervention strategy was not significantly associated with differences in *Giardia* prevalence at any test point.

Estimated Costs of Control Strategies

Our survey of parents of *Giardia*-infected children at the time of intervention found the mean cost of physician visits associated with this diagnosis to be $34 (range $1–$120) and the mean cost of medication to be $12 (range $2–$60). Forty-two percent of these parents reported missing work in association with their child's exclusion from day care, with a projected mean of 6.6 days for parents in Group 1, and 3.5 days for parents in Groups 2 and 3.

Table 5 presents the projected estimates of costs which result from applying these results to each of the intervention groups at the time of intervention and throughout the six months of study including the intervention. These estimates are based on the actual number of symptomatic and asymptomatic children detected at the time of intervention, and on the additional cases of *Giardia*-positive diarrhea detected during follow-up. For the initial intervention, Group 2 had substantially lower projected costs than Group 1 in terms of both medical costs and of day care and parent work days lost. Group 3 had lost time costs similar to Group 2, but had substantially higher medical costs resulting from the treatment of asymptomatic as well as symptomatic children.

TABLE 5—Estimated Costs of *Giardia* Control Strategies in Study Day Care Centers at the Time of Initial Intervention and Total during Six Months of Observation, by Intervention Group

	Cost at Initial Intervention/Total Cost Over Six Months		
	Group 1	Group 2	Group 3
No. of children excluded	52/58	22/51	18/34
Medical treatments required	52/58	22/51	67/83
DCC days lost	20/580	110/255	90/170
Parent work days lost	114/161	32/75	26/50
Physician costs	$1,768/$1,972	$748/$1,734	$2,278/$2,822
Prescription costs	$624/$696	$264/$612	$804/$996

By the end of six months, the large number of cases of *Giardia*-positive diarrhea in Group 2 resulted in a total number of excluded children, and resulting projected medical costs, similar to those of Group 1. Because of the difference in exclusion requirements between Group 1 and the other two groups, the projected numbers of day care and work days lost remained substantially lower in Groups 2 and 3 throughout the period of study.

Discussion

Strategies for control of specific infectious diseases in child day care must be principally based on concern for the health of the infected or ill child, the probability of transmission of infection to other children or adults, the effect of infection on these other persons, and the preventive or curative control measures available. At the same time, these strategies must be developed and implemented taking into account their impact on the child, the family, employers, and day care providers. Physicians and local health authorities have usually had to develop disease control interventions in the absence of adequate information regarding their effectiveness and impact.[6-8]

Giardia lamblia has been recognized as a problem in day care of diaper age children, and *Giardia* infection has been associated with day care center attendance.[1,2,9,10] In Phoenix and in some other day care environments, asymptomatic *Giardia* infection among infants and toddlers in centers has also been documented to be prevalent.[4,5] These asymptomatic infected children present a special disease control dilemma, since limited data suggest no adverse nutritional impact of such infection among well-nourished immunocompetent children,[5] and no increase in diarrhea rate among such children compared with *Giardia*-negative children in the same settings.[4,5] However, these asymptomatic infected children do represent a potential source of infection to others.

The existence of a very strict health department strategy for control of *Giardia lamblia* in Maricopa County (greater Phoenix) day care centers gave us the opportunity to perform this randomized controlled evaluation of that strategy in comparison with two less strict strategies. Our study was designed as an effectiveness evaluation of these intervention strategies under existing conditions of day care centers, turnover of enrollment, and medical treatment provided by community care providers, rather than an efficacy evaluation in which these variables would be controlled by the investigators. For the same reason, we continued to identify day care centers having *Giardia*-infected infants and toddlers through the existing health department surveillance mechanism and continued the health department policy of identifying *Giardia* through a single stool examination.

Several immediate differences emerged between intervention groups. At one month after intervention, significant reductions in *Giardia* prevalences existed in the two groups of centers where both symptomatic and asymptomatic children were treated; a lesser non-significant reduction in prevalence occurred in the group of centers where only symptomatic children were treated. Part of this immediate reduction in *Giardia* prevalence was attributable to *Giardia*-positive children leaving the study centers. Although we did not investigate whether these *Giardia*-positive children moved to other day care centers and infected other children, molecular epidemiological study of *Shigella*-infected day care center children has identified such transfer of infection to other child groups in response to control measures.[11]

At the end of the six-month follow-up period, no control strategy was associated with significantly lower prevalences of *Giardia* among infants and toddlers in study centers, although the six-month prevalences in all three groups were significantly lower than the prevalences at the time of intervention. Of equal importance is the recognition that the 8 percent overall prevalence of *Giardia* at six months after intervention is essentially the same as the 10.5 percent prevalence of *Giardia* among the infants and toddlers who entered study centers during the follow-up period; the majority of these new enrollees had not previously attended a day care center. *Giardia*-positive new enrollees accounted for almost 40 percent of *Giardia* infection at six months. The unexpectedly high prevalence of *Giardia* among Phoenix infants and toddlers without previous day care histories, and the high turnover of children characteristic of this center population, suggests that *Giardia* control strategies for Phoenix day care centers cannot expect to reduce prevalence below this apparent background level.

These characteristics of the Phoenix population of infant-toddler center users probably limit the external validity of our findings to similar populations. It may be that in an infant-toddler population with very low prevalences of *Giardia* and low rates of center turnover, strict control strategies might result in elimination of *Giardia* from a given center. In the Phoenix day care center environment, however, our data indicate that the existing strategy did not achieve substantially greater control of *Giardia* infection, especially in comparison with the strategy that treated both symptomatic and asymptomatic infection with less stringent exclusion requirements.

Our data do not permit general recommendations regarding the need for *Giardia* testing of young children in day care who experience diarrhea. They do suggest that, in environments comparable to Maricopa County, *Giardia* testing should be considered if microbiological testing is indicated in an episode of diarrhea in an infant or toddler (whether in a day care center or not) or if a day care center is experiencing a diarrhea outbreak.

Our projected estimates of the cost of the different strategies were consistent with parents' and day care owners' experience that the existing strategy resulted in substantial numbers of child day care and parent work days lost. While initially the least costly, Group 2 experienced more diarrhea during follow-up, resulting in total medical costs comparable to the existing strategy.

Our data do not permit distinction of diarrhea caused by *Giardia* infection from diarrhea of other etiology occurring in a child who was coincidentally infected by *Giardia*. However, if the reduced initial costs of Group 2 were accomplished at the expense of additional preventable child illness, this would clearly be an unacceptable strategy. The greatest difference in projected costs between strategies, and that most important to parents and day care operators, resulted from the difference in exclusion requirements. The *Giardia* prevalence results and diarrhea experience in Group 3 suggest that these less stringent requirements did not result in an outcome substantially different from that of the stricter exclusion policy.

Although the cost of conducting controlled trials of public health disease control strategies restricts the usefulness of this approach, documentation to the extent possible of the effects and impact of these strategies can represent an important contribution to day care disease control efforts. Certainly, in various child day care environments additional information is needed on the control of *Giardia lamblia* and other infectious diseases important in preschool age children. □

Acknowledgments

The authors wish to express their appreciation to the parents, children, and staff of the day care centers who collaborated in this study, to the study nurses, and to H. Robert Baird, MS, for his support in data analysis. This work was supported by a grant from the Arizona Disease Control Research Commission. Presented in part at the 99th Annual Meeting of the Society for Pediatric Research, Washington DC, May 1989.

References

1. Black RE, Dykes AC, Sinclair SP, Wells JG: Giardiasis in day care centers: Evidence of person-to-person transmission. *Pediatrics*. 1977;60:486–491.
2. Claquin P, White M, Schultz MG, Juranek DD: Diarrheal illness in day care centers, Tucson, Arizona. Atlanta: Centers for Disease Control, Report EPI-80-1-2, April 1981.
3. Pickering LK, Evans DG, DuPont HL, Vollet JJ III, Evans DJ Jr: Diarrhea caused by *Shigella*, rotavirus, and *Giardia* in day care centers: Prospective study. *J Pediatr.* 1981;99:51–56.
4. Bartlett AV, Moore MD, Gary GW, Starko KM, Erben JJ, Meredith BA: Diarrheal illness among infants and toddlers in day care centers: I. Epidemiology and pathogens. *J Pediatr.* 1985;107:495–502.
5. Pickering LK, Woodward WE, DuPont HL, Sullivan P: Occurrence of *Giardia lamblia* in children in day care centers. *J Pediatr.* 1984;104:522–526.
6. The Child Day Care Infectious Diseases Study Group, US Centers for Disease Control: Public health considerations of infectious diseases in child day care centers. *J Pediatr.* 1984;105:683–701.
7. Klein JO: Infectious diseases and day care. *In:* Osterholm MT, *et al*, (eds): Infectious Diseases and Child Day Care: Management and Prevention. Chicago: University of Chicago Press, 1986.
8. Shapiro ED, Kuritsky J, Potter J: Policies for the exclusion of ill children from group day care: An unresolved dilemma. *In:* Osterholm MT, *et al*, (eds): Infectious Diseases and Child Day Care: Management and Prevention. Chicago: University of Chicago Press, 1986.
9. Sealy DP, Schuman SH: Endemic giardiasis and day care. *Pediatrics.* 1983;72:154–158.
10. Novotny TE, Hopkins RS, Shillam P, Janoff EN: Prevalence of *Giardia lamblia* and risk factors for infection among children attending day care facilities in Denver. *Public Health Rep.* 1990;105:72–75.
11. Tacket CO, Cohen ML: Shigellosis in day care centers: Use of plasmid analysis to assess control measures. *Pediatr Infect Dis.* 1983;2:127–129.

Characteristics of Maternal Employment during Pregnancy: Effects on Low Birthweight

ABSTRACT

Background: Although maternal employment is considered a risk factor for low birthweight (LBW), the manner in which employment might affect birthweight is poorly understood. In this analysis, selected charateristics of employment during pregnancy were examined for effects on pregnancy outcomes.

Methods: Work characteristics included the number of hours per week, physical activities, and environmental conditions. The outcomes of interest were fetal growth retardation (<2500 grams at term) and preterm delivery (<37 weeks). The study population consisted of 2711 non-Black, married mothers who participated in the 1980 National Natality Survey (NNS). The NNS data were merged with data from the 1977 revision of *The Dictionary of Occupational Titles* (DOT) from which measures of occupational physical activities and environmental exposures were obtained. Logistic regression was used in the analysis.

Results: Those who worked 40 or more hours per week were more likely than women who worked fewer hours to have a low birthweight delivery at ≥37 weeks. No physical or environmental characterics of work were associated with low birthweight or preterm delivery.

Conclusions. Non-Black married American women may face a risk of delivering low-birthweight babies at or near term only if they work 40 or more hours each week. However, the lack of risk associated with other characteristics of work may be a function of measurement error in the DOT data source or of low levels of exposure in the analysis population. (*Am J Public Health*. 1991;81;1007–1012)

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Introduction

Infants weighing 2500 g or less at birth may be small for gestational age, the result of a preterm birth, or both.[1] The two low-birthweight (LBW) categories share many risk factors[2] but are caused by different physiological mechanisms.[3] For women who work during pregnancy, characteristics of employment have been associated inconsistently with both outcomes.

Studies in developing countries suggest that hard physical labor during pregnancy has adverse effects on fetal growth.[4–6] In industrialized nations, standing,[7–10] carrying or lifting heavy loads,[8,11] strenuous physical effort,[12–15] work on assembly lines[12] and industrial machines,[13] and some occupations that involve physical effort, such as chambermaid, janitor, and hospital worker,[8,16] have been associated with delayed fetal growth, preterm birth, or both. However, in other investigations, each of these findings has either not been supported[8,12,17–22] or has not been examined.

Studies of environmental conditions such as temperature, dust, and moisture have found few associations with either category of LBW.[8,17,23] However, one study found loud noises associated with lower birthweight,[8] while in another, an "environment" index was associated with preterm birth.[13]

Physical and environmental exposures at work may influence pregnancy by decreasing uterine blood flow,[24,25] by stimulating production of catecholamines[26,27] or both. Presumably, these processes can be accelerated or decelerated by amount of exposure. Hence, studies in which number of hours worked per day or week are taken into account are potentially informative. However, the studies that have included these variables have produced inconsistent results.[7,8,12,13,18,19,22,28]

Most of the research in this area has been done in Europe,[10–13,19,20,29] with some recent reports from Montreal.[8,16] Of the 10 American studies reported in the literature,[7,9,14,15,17–19,22,28,30] four found no significant effects of maternal employment.[17–19,28] Also, the analysis of the Collaborative Perinatal Project data of the early 1960s[7] may not be applicable to current working populations. Most of the US studies have focused on a few physical activities (notably that of standing) and on hours and duration of work to the exclusion of environmental exposures. Also, confidence in the findings of the studies of American women is tempered by measurement problems. In some cases, employment characteristics were assigned post hoc by the investigators or other experts to occupational titles.[7,9,17,18] Variations by individual or work site were not taken into account, and the validity of the coding schemes was unknown. Another approach was to collect employment data after the delivery,[19,22] when recall may

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This paper was submitted to the journal November 8, 1990, and accepted with revisions April 5, 1991.

Editor's Note. See related editorial on p. 973.

have been clouded by elapsed time, the relative significance of the birth event, the health of the infant, or all of these factors. Homer et al.[14,30] avoided these sources of error by imputing employment characteristics from an independent data base. However, they may have introduced a new source of self-report bias because the data were collected by surveying workers.

The purpose of the present study was to examine the effects of selected physical and environmental characteristics of maternal employment and amount of work (hours/week) on fetal growth and preterm delivery in a representative sample of contemporary American women. To avoid the measurement error in assessment of job characteristics associated with self-report and expert opinion, data on employment characteristics were obtained from an independent source derived primarily from work-site observations.

Methods

Data Sources

This study is an analysis of the 1980 National Natality Survey (NNS), a follow-back survey of a probability sample of registered births in the United States. Approximately 1 in 400 live births ≥2500 g and 1 in 95 live births <2500 g are in the NNS sample, which totals 9941. Data were collected from birth certificates and requested by mail from hospitals and other health care providers. In addition, detailed sociodemographic and behavioral data as well as basic information on employment during the 12 months before the birth were requested by mail from mothers who were married at the time of delivery. The data collection process involved three mailings and an attempt to interview nonrespondents by telephone. Overall response was 79.5% from married mothers and 76.1% from hospitals. All data were collected retrospectively, within 1 year of the 1980 deliveries.[31,32]

The NNS data set was merged with the 1977 Dictionary of Occupational Titles (DOT), which includes descriptive data on more than 12 000 distinct occupations in more than 75 000 sites.[33] DOT data were collected by on-site interviews with workers and from observations of jobs as they were performed.[34] A detailed job analysis schedule[33] was completed for each job and, if feasible, verified by the worker's immediate supervisor.[34] One or two workers were observed for each job analysis. Scores on 44 work characteristics were then assigned by the analyst based on detailed guidelines and examples.[35]

Study Population

The study population consisted of 2711 non-Black (97% White) singleton live births to married mothers who worked during pregnancy and provided sufficient data on occupation and industry to match with DOT job characteristics. NNS subjects with imputed data entries on employment items, birthweight, or gestational age at delivery were not included in the 2711. The population was limited to married women because employment data were available only from the mothers' questionnaire. This restriction caused the number of Black mothers eligible for analysis to be so small (n = 399) that they were excluded from the analysis.

Two sets of analyses were conducted to distinguish between the effects of work characteristics on fetal growth and on gestational age, since the two conditions are due to different physiologic processes.[3] For analyses in which term low birthweight was the dependent variable, infants who were clearly preterm (<37 weeks) were removed from the analysis population. Similarly, in analyses of preterm births, infants who were low birthweight (fetal growth retarded) at term (≥37 weeks) were removed. The purpose of this was to exclude infants with identifiable morbidities from the groups to which low birthweight and preterm births were compared. In addition, women who started work after the eighth gestational month were excluded from analyses of preterm births.

Derivation of the analysis samples is show schematically in the Appendix. Each exclusion produced more homogeneous populations with lower risk of adverse pregnancy outcomes. In general, the analysis samples were of lower parity, had more education, received more prenatal care, and gained more weight that the exclusion groups.

Variables

The dependent variables, low birthweight (<2500, ≥2500 g) and preterm delivery (<37, ≥37 weeks), were constructed from the birth certificate source in the NNS. Number of hours worked per week in a company, business or organization was taken from the NNS mothers' questionnaire and categorized as 1 to 20, 21 to 39, and ≥40. Three measures of physical activity were constructed from DOT data. Physical strength was derived from field observations of standing, walking, sitting, lifting, carrying, pushing, and pulling objects; jobs were categorized as sedentary, light, medium, heavy, or very heavy based on these activities.[35] The other physical activities were climbing/balancing and stooping/kneeling/crouching/crawling. A score of 1 for presence or 0 for absence was recorded for each DOT job on these variables.[35]

Six DOT characteristics were used as measures of environmental conditions: (1) work performed outdoors without effective protection from the weather; (2) extreme cold with or without abrupt variations in temperature; (3) extreme heat with or without abrupt variations in temperature; (4) constant or intermittent loud noises; (5) physical hazards (e.g., work on scaffolding, exposure to electrical shock, exposure to toxic chemicals); and (6) elements in the atmosphere (i.e., fumes, odors, dusts, mists, gases, or poor ventilation.) These characteristics were also scored 1 or 0, as above.[35]

DOT job titles do not correspond exactly with the NNS census codes for occupation and industry. To merge the two files, a bridge tape created for this purpose was used. Each DOT value on the bridge tape represented the level of the characteristic for incumbents of a given occupation in the 1971 Current Population Survey. Levels for the physical exertion variable were population means, while values for the remaining DOT characteristics were percentages (of the population in the given occupation with the characteristic present).[36,37]

In this analysis, the scores described above were categorized into low, medium, and high groups, based on distribution in the NNS population. For symmetrical distributions, the lowest and highest deciles were used to designate low and high groups. In cases of asymmetrical distributions with long tails caused by large percentages of jobs in the population that did or did not have the particular DOT characteristic, cut points at the tails were used to designate low or high categories.

Six NNS variables were considered potential confounders.[2] Maternal age, parity, education, and adequacy of prenatal care[38] were categorical variables constructed from birth certificate data. Number of cigarettes smoked per day after confirmation of pregnancy was a continuous variable derived from the mothers' questionnaire. Prepregnant weight adjusted for height was based on data from hospital and mothers' questionnaires. To construct the variable, the National Center for Health Statistics (NCHS) formula, weight/height$^{1.5}$ was applied to the population of all working women.[39] Adjusted weights were categorized as lean (≤14 percentile), normal (15 to 84 percentile), and overweight (≥85 percentile).

Pregnancy complications and weight gain were available in the NNS, but these variables were not controlled in the analysis because effects of work on fetal growth and gestational duration may occur through these conditions. Other potential confounders such as family income, wantedness of pregnancy, and interval since last pregnancy were not used because of missing data. Indicators of previous LBW or preterm birth were not available.

Data Quality

Cases with missing or imputed entries for birthweight, gestational age, and all characteristics of employment were excluded from the analysis. Fewer than 1% of missing data were imputed for the other variables, with one exception, parity at 13.1%. The imputation process and other NNS editing and cleanup processes are described elsewhere.[31,32] Birthweight and gestational age combinations were examined for plausibility according to David's criteria.[40] Forty-five subjects had implausible combinations and were excluded from the analysis. NNS occupation and industry combinations were compared with DOT job characteristics to assess face validity. The investigators concluded that correspondence between the two data sets was acceptable.

Analysis

Analysis was performed by multiple logistic regression using sampling weights. Low-birthweight infants were oversampled in the NNS, and there was differential reporting by age and birth order, resulting in unequal probabilities of selection.[41] Sampling weights were assigned to each birth in each stratum, defined by birthweight, age, and birth order. The analyses reported here involved computing weighted means and proportions and then constructing logistic regression models using the weights. The analysis was conducted using the software PROC RTI LOGIT written for fitting models to complex survey data.[42] This procedure incorporates sampling weights into the estimation of the logistic regression parameters.

Several logistic regression models were constructed with low birthweight as well as preterm delivery as dependent variables. In addition to the work characteristics, the six potential confounders were used as independent variables. Some models included interaction terms for the DOT characteristics by number of hours worked and parity. Generally, a *P* value of .05 was used to designate significance of the F tests of each parameter. Odds ratios and 95% confidence intervals (CI) were also calculated.

Findings

The study population (Table 1) had relatively low percentages of low birthweights, preterm births, and maternal risk characteristics. Each of the analysis populations had similar distributions.

In the population who delivered at term (Table 2), the percent LBW increased directly with number of hours worked, but the only DOT variable distributed in the hypothesized direction was extreme cold. When the effects of confounders were controlled, only number of hours worked was statistically significant. With 1 to 20 hours as the standard, the odds of having a LBW infant were 1.4 (CI = 0.80–2.36) times greater for women who worked 21 to 39 hours, and 1.7 (CI = 1.03–2.68) times greater for women who worked ≥40 hours (Table 3).

Among women who started work at 8 or fewer months of gestation and had infants who were not LBW at term (Table 4), there is no discernible pattern in the distribution of preterm births by number of hours worked or by the DOT characteristics, with one exception: extreme cold. None of the employment characteristics was significant when confounding variables were controlled in logistic regression analysis.

Discussion

The findings suggest that working long hours during pregnancy is associated with low-birthweight deliveries at 37 or more weeks' gestation for married, non-Black women. None of the other characteristics of maternal employment are strongly related to low birthweight at term or to preterm delivery in this population.

The relationship between working long hours and delivery of a full-term, low-birthweight infant is difficult to interpret in light of the other, null, results. The relationship held when each of the other characteristics of work was included in the model, suggesting that long working hours has a strong independent effect on low birthweight. Working 40 or more hours per week may reflect elements of psychological stress or physical fatigue that were not represented in the other variables. Unlike the dramatic findings of Mamelle, et al.[13], analysis of interactions between hours worked and the other work characteristics produced no additional information. However, because interaction analyses were sometimes based on few observations in the combined categories of interest, the possibility cannot be ruled out that working long hours in jobs with identifiable characteristics is the influential factor.

TABLE 1—Distribution of Pregnancy Outcomes and Potential Confounders for 2711 Non-Black Married Women Who Worked during Pregnancy

Characteristic	Percentage of the Population^a
Birthweight (g)	
<2500	4.2
≥2500	95.8
Gestational age (weeks)	
<37	6.8
≥37	93.2
Maternal age (years)	
<20	6.7
20–24	34.8
25–29	37.7
≥30	20.9
Parity	
0	48.6
1–3	48.3
≥4	3.2
Education (years)	
<12	7.7
12	44.6
>12	47.7
Prenatal care	
Adequate	79.8
Less than adequate	20.2
Prepregnant weight standardized by height	
Lean	16.2
Normal	74.8
Overweight	8.9
Number of cigarettes smoked per day since pregnancy confirmed	
0	75.5
1–9	7.3
10–19	8.5
≥20	8.6

^{a}Weighted for differential probabilities of selection. The sums of percentages may not add to 100 because of rounding.

Some measurement issues also must be considered in interpreting this finding. One is the retrospective nature of the NNS data. In the year following the 1980 birth, respondents probably would have provided accurate information about occupation and industry, but recall of the number of hours worked per week may

TABLE 2—Distribution of Characteristics of Work in the Analysis Population and Percent Low Birthweight in Each Category, 2379 Infants of Non-Black Women Who Delivered at Term (≥37 weeks) and Worked during Pregnancy

Characteristic of Work	Percentage in the Population[a]	Percentage Weighing <2500 g[a]
Hours worked/week		
1–20	16.5	1.4
21–39	26.1	1.7
≥40	57.4	2.0
Strength		
Low	11.4	2.8
Medium	78.9	1.7
High	9.7	1.9
Climbing/balancing		
Low	41.2	1.7
Medium	47.1	2.2
High	11.7	1.1
Stooping		
Low	14.9	1.7
Medium	75.5	1.9
High	9.6	1.4
Work performed outdoors		
Low	67.6	1.9
Medium	26.6	1.7
High	5.8	1.6
Extreme cold		
Low	84.7	1.8
Medium	10.7	1.7
High	4.6	2.7
Extreme heat		
Low	66.3	1.7
Medium	24.3	2.4
High	9.4	1.8
Loud noises		
Low	32.4	1.8
Medium	63.0	1.9
High	4.6	2.1
Physical hazards		
Low	37.3	1.8
Medium	53.4	1.9
High	9.3	1.9
Atmospheric elements		
Low	65.1	1.9
Medium	24.7	1.9
High	10.2	1.5
Total	100.0	1.9

[a] Weighted for differential probabilities of selection.

have been blurred by time and subsequent events. Also, the number of hours worked per week could have changed during pregnancy, but the NNS questionnaire did not capture this dynamic aspect. Employment data were limited to the mother's chief job. Some women may have worked additional jobs that could have extended the number of hours worked per week. The effects of these deficiencies on the findings would be underestimation of the coefficients of hours worked.

The null findings from analysis of DOT physical and environmental factors should be considered from three perspectives. First, these results are consistent with other studies of American populations,[17–19,22,28] but they contrast sharply with findings from major studies in France[11–13] and Canada.[8,16] Perhaps the working conditions of US women are more favorable for birthweight and length of gestation than those in other industrialized countries, or perhaps the relatively advantaged and homogeneous character of our sample is not comparable to the diverse populations in the French and Canadian studies. Also, the quality of the DOT data (discussed below) may have been inferior to data collected directly, albeit retrospectively, from working women in the French and Canadian studies.

A second possible interpretation is derived from the fact that the low-risk status of the population may have diminished our ability to detect effects of physical and environmental exposures within the sample. Because the study population was sociodemographically advantaged, they may have tolerated physical and environmental stresses of work better than a less advantaged group. Or they may have undertaken jobs with fewer physical demands and more comfortable working environments than less advantaged women. Another analysis of the 1980 NNS data found that advantaged women tended to work in the professional, technical, and managerial sector,[43] where physical and environmental insults are likely to be few. We also found evidence that the study population had some control over work-related exposures. There were considerably higher percentages of women who stopped working in the first 3 months of pregnancy who had jobs categorized as high on each of the DOT factors. This tendency was also found, although not as consistently, for women who worked short (1 to 20) hours.

A third possibility, misclassification bias in DOT data, warrants close attention. The only positive result in this study was based on NNS data, whereas all analyses of DOT variables produced null results. We selected the DOT as our source of employment data because, as an independent source derived primarily from on-site observations, it allowed us to avoid self-report and expert opinion biases in job measurement. The DOT has been used extensively for research[44] and has been shown to correlate reasonably well with another large national employment data base.[45] Linking of macrolevel data bases with individual data is advocated as an informative tool for initial epidemiologic studies. However, an inherent bias toward the null of coefficients derived from multiple regression analysis has been reported.[45] In addition, a number of documentation and procedural problems with data collection and assignment of scores in the DOT have been identified.[34] These problems could have resulted in misclassification of the variables of interest. We attempted to identify misclassification by examining DOT values in each occupation or industry for face validity before any analyses were conducted. Also, after analyses of physical and environmental variables were completed, we created three separate measures of high vs low risk for physical exertion from NNS occupation data only, and repeated the logistic regression analysis with each. Consistent with our DOT findings, none of these measures was associated with either of the outcomes. (The definitions of these variables

TABLE 3—Results of Multiple Logistic Regression Analysis for Non-Black Women Who Worked during Pregnancy and Delivered at 37 or More Weeks' Gestation

Variable	Beta	Standard Error	F[a]	*P*	OR[b]	95% Confidence Interval
Maternal age (years)						
<20	0.088	0.069	1.59	.207	0.9	0.80–1.05
≥30	0.253	0.056	20.37	.000	1.3	1.15–1.43
Number of cigarettes smoked per day	0.048	0.007	46.51	.000	1.1	1.03–1.06
Prenatal care less than adequate	0.4756	0.176	7.32	.007	1.6	1.14–2.27
Hours worked						
21–39	0.319	0.275	1.35	.245	1.4	0.80–2.36
≥40	0.508	0.245	4.31	.038	1.7	1.03–2.68

[a] F tests are with (1, α) *df.*
[b] OR = odds ratio.
Note: Test for overall model: F 12.60; *df* 6; *P* < 0.0001.

and results of the analyses are available from the authors upon request.)

The promise for statistical control of potential confounders in the NNS was substantial, but because of missing data, several critical variables such as income, pregnancy interval, and alcohol consumption were not used. Other key variables (e.g., previous LBW, previous pregnancy complications, psychosocial stress, and physical activity outside of work) were not available. While it is unlikely that control of these factors would have altered the null results, they should be included in future research.

In light of the preceding observations, it is prudent to conclude that non-Black, married American women may face a risk of delivering low-birthweight babies at or near term only if they work 40 or more hours each week. However, the lack of risk associated with other characteristics of work may be a function of measurement error in the DOT data source or of low levels of exposure in this sociodemographically advantaged population. With regard to the potential effects of physical and environmental characteristics in the workplace, this study has taken the logical next step in a progression of epidemiologic investigations that includes retrospective self-reports of job exposures, post hoc assignment of exposures by expert panels, and merging of macrolevel job characteristic files with individual-level data. It is now clear that these less expensive approaches to studying maternal employment and pregnancy outcome are not sufficient. Studies based on high-quality data collected prospectively from the women themselves are imperative. □

Acknowledgments

The authors wish to thank Drs. Paul Placek and Ken Keppel of the National Center for Health Statistics for their advice and guidance concerning the 1980 NNS, and Dr. Ken Spenner for consultation on the DOT. Appreciation is also extended to Drs. J. Richard Udry, Karen Gleiter, David Savitz, and Milton Kotelchuck for assistance at several points during the study, to Susan Harris for typing multiple drafts of this paper with characteristic skill and good humor, and to John Feaganes for programming assistance. This research was supported by Grant No. R01-HD-22150 from the National Center for Child Health and Human Development.

The manuscript is based in part on a paper presented at the 116th Annual Meeting of the American Public Health Association, November 1988, Boston, Mass.

TABLE 4—Distribution of Characteristics of Work in the Analysis Population and Percentage of Preterm Deliveries in Each Category for 2511 Infants of Non-Black Women Who Started Work at 8 or Fewer Months' Gestation[a]

Characteristic of Work	Percentage in the Population[b]	Percentage of <37-Week Deliveries[b]
Hours worked/week		
1–20	16.4	6.4
21–39	26.2	7.6
≥40	57.4	6.9
Strength		
Low	11.4	7.9
Medium	78.7	6.6
High	9.9	8.9
Climbing/balancing		
Low	41.3	7.1
Medium	46.9	6.9
High	11.7	6.9
Stooping		
Low	14.7	6.1
Medium	75.7	7.2
High	9.6	6.9
Work performed outdoors		
Low	67.4	7.1
Medium	26.8	7.2
High	5.6	5.1
Extreme cold		
Low	84.5	6.8
Medium	10.7	6.8
High	4.8	11.1
Extreme heat		
Low	66.2	7.0
Medium	24.4	7.2
High	9.4	6.4
Loud noises		
Low	32.7	6.3
Medium	62.7	6.3
High	4.5	7.1
Physical hazards		
Low	37.2	6.9
Medium	53.7	7.2
High	9.2	6.0
Atmospheric elements		
Low	64.9	6.7
Medium	25.0	8.1
High	10.1	6.0
Total	100.0	7.0

[a] 1 month = approximately 4 weeks.
[b] Weighted for differential probabilities of selection.

References

1. Institute of Medicine. *Preventing Low Birthweight.* Washington, DC: National Academy Press; 1985.
2. Kramer MS. Intrauterine growth and gestational duration determinants. *Pediatrics.* 1987;80:502–511.
3. Bragonier JR, Cushner IM, Hobel CJ. Social and personal factors in the etiology of preterm birth. *In:* Fuchs F, Stubblefield PG, eds. *Preterm Birth: Causes, Prevention and Management.* New York: Macmillan Publishing Co; 1983: chap 4.
4. Tafari N, Naeye RL, Gobezie A. Effects of maternal undernutrition and heavy physical work during pregnancy on birth weight. *Br J Obstet Gynaecol.* 1980;87:222–226.
5. Manshade J, Eckels R, Manshade-Desmit U, Vlietinck R. Rest versus heavy work during the last weeks of pregnancy: influence on fetal growth. *Br J Obstet Gynaecol.* 1987;94:1059–1067.
6. Launer L, Villar J, Kestler E, DeOnis M. The effect of maternal work on fetal growth and duration of pregnancy: a prospective study. *Br J Obstet Gynaecol.* 1990;97:62–70.
7. Naeye R, Peters E. Working during pregnancy: effects on the fetus. *Pediatrics.* 1982;69:724–727.
8. McDonald AD, McDonald JC, Armstrong B, Cherry NM, Nolin AD, Robert D. Prematurity and work in pregnancy. *Br J Ind Med.* 1988;45:56–62.

9. Teitelman AM, Welch LS, Hellenbrand KG, Bracken MB. Effect of maternal work activity on preterm birth and low birthweight. *Am J Epidemiol.* 1990;131:104–113.
10. Nurminen T, Lusa S, Ilmarinen J, Kurppa K. Physical work load, fetal development and course of pregnancy. *Scand J Work Environ Health.* 1989;15:404–414.
11. Saurel-Cubizolles M, Kaminski M, Llado-Arkhipoff J, et al: Pregnancy and its outcome among hospital personnel according to occupation and working conditions. *J. Epidemiol Community Health.* 1985; 39:129–134.
12. Saurel-Cubizolles M, Kaminski M. Pregnant women's working conditions and their changes during pregnancy. *Br J Ind Med.* 1987;44:236–243.
13. Mamelle N, Laumon B, Lazar P. Prematurity and occupational activity during pregnancy. *Am J Epidemiol.* 1984; 119:309–322.
14. Homer CJ, Beresford SA, James SA, Siegel E, Wilcox S. Work-related physical exertion and risk of preterm, low birthweight delivery. *Paediatr Perinat Epidemiol.* 1990;4:161–174.
15. Ramirez G, Grimes RM, Annegers JF, Davis BR, Slater CH. Occupational physical activity and other risk factors for preterm birth among US Army primigravidas. *Am J Public Health.* 1990;80:728–729.
16. McDonald AD, McDonald JC, Armstrong B, et al. Occupation and pregnancy outcome. *Br J Ind Med.* 1987;44:521–526.
17. Meyer B, Daling J. Activity level of mother's usual occupation and low infant birth weight. *J Occup Med.* 1985;27:841–847.
18. Zuckerman B, Frank D, Hingson R, Morelock S, Kayne H. Impact of maternal work outside the home during pregnancy on neonatal outcome. *Pediatrics.* 1986; 77:459–464.
19. Berkowitz R. Physical activity and the risk of spontaneous preterm delivery. *Reprod Med.* 1983;28:581–588.
20. Rabkin CS, Anderson HR, Bland JM, Brooke OG, Chamberlain G, Peacock JL. Maternal activity and birthweight: a prospective population-based study. *Am J Epidemiol.* 1990;131:522–531.
21. Najman JM, Morrison J, Williams GM, Andersen MJ, Keeping JD. The employment of mothers and the outcomes of their pregnancies: an Australian study. *Public Health.* 1989;103:189–198.
22. Klebanoff MA, Shiono PH, Rhoads GG. Outcomes of pregnancy in a national sample of resident physicians. *N Engl J Med.* 1990;323:1040–1045.
23. Hartikainen-Sori A, Sori M. Occupational and sociomedical factors in preterm birth. *Obstet Gynecol.* 1989;74:13–16.
24. Easterling TR, Schmucker BC, Benedetti TJ. The hemodynamic effects of orthostatic stress during pregnancy. *Obstet Gynecol.* 1988;72:550–552.
25. Hagan RD, Diaz FJ, Horvath SM. Plasma volume changes with movement to supine and standing positions. *J Appl Physiol.* 1978;45:414–417.
26. Timio M, Gentili S, Pede S. Free adrenaline and noradrenaline excretion related to occupational stress. *Br Heart J.* 1979; 42:471–474.
27. Schnider SM, Wright RG, Levinson G, et al. Uterine blood flow and plasma norepinephrine changes during maternal stress in the pregnant ewe. *Anesthesiology.* 1979; 50:524–527.
28. Marbury M, Linn S, Monson R, et al. Work and pregnancy. *J Occup Med.* 1984; 26:415–421.
29. Ahlborg G, Bodin L, Hagstedt C. Heavy lifting during pregnancy: a hazard to the fetus? A prospective study. *Int J Epidemiol.* 1990;19:90–97.
30. Homer CJ, James SA, Siegel E. Work-related psychosocial stress and risk of preterm, low birthweight delivery. *Am J Public Health.* 1990;80:173–177.
31. Placek PJ. The 1980 National Natality Survey and National Fetal Mortality Survey: methods used and PHS agency participation. *Public Health Rep.* 1984;99:111–116.
32. Keppel KG, Heuser RL, Placek PJ, et al. *Methods and response characteristics: 1980 National Natality and Fetal Mortality Surveys.* Hyattsville, Md: Public Health Service, National Center for Health Statistics; September 1986. DHHS Publication (PHS) 86-1374.
33. Cain PS, Treiman DJ. The Dictionary of Occupational Titles as a source of occupational data. *Am Soc Rev.* 1981;46:253–278.
34. Miller AR, Treiman DJ, Cain PS, Roos PA, eds. *Work, Jobs, and Occupations: A Critical Review of the Dictionary of Occupational Titles.* Washington, DC: National Academy Press; 1980.
35. US Department of Labor: *Handbook for Analyzing Jobs.* Washington, DC: US Government Printing Office; 1972.
36. US Department of Labor. *DOT Data Tape Documentation.* Washington, DC; 1977.
37. Spenner KI. Occupational characteristics and classification systems: new uses of the Dictionary of Occupational Titles in social research. *Soc Meth Res.* 1980;9:239–264.
38. Kessner DM, Singer J, Kalk CE, Schlesinger ER. *Infant Death: An Analysis by Maternal Risk and Health Care.* Washington, DC: Institute of Medicine; 1973.
39. National Center for Health Statistics. *Obese and Overweight Adults in the US.* Hyattsville, Md.; 1983. Vital and Health Statistics, National Health Survey, Ser 11, No. 230.
40. David RJ. The quality and completeness of birthweight and gestational age data in computerized birth files. *Am J Public Health.* 1980;70:964–970.
41. National Center for Health Statistics. *Public Use Data Tape Documentation: National Natality Survey 1980, National Fetal Mortality Survey 1980.* Hyattsville, Md: Public Health Service; December 1983.
42. Shah BV, Folsom RE, Harrell FE, Dillard CN. *Survey Data Analysis Software for Logistic Regression.* Research Triangle Park, NC: Research Triangle Institute; 1984. Subcontract No. A-3097 (8149)-293.
43. Savitz DA, Whelan EA, Rowland A, Kleckner RC. Maternal employment and reproductive risk factors. *Am J Epidemiol.* 1990;132:933–945.
44. Price J. Annotated bibliography of research uses of the Dictionary of Occupational Titles. *In:* Miller AR, Treiman DJ, Cain PS, Roos PA, eds. *Work, Jobs, and Occupations: A Critical Review of the Dictionary of Occupational Titles.* Washington, DC: National Academy Press; 1980.
45. Schwartz JE, Piper CF, Karasek RA. A procedure for linking psychosocial job characteristics data to health surveys. *Am J Public Health.* 1988;78:904–909.

APPENDIX—Derivation of Analysis Samples National Natality Survey, 1980

Total NNS Sample	9941
Married Subjects	7825
Singleton Live Births	7514
Mother Workded During Pregnancy	4532
Non-Black Live Births	4133
Matched DOT	3333
Complete Data on Work and Outcome Variables	2711

LBW Analysis Sample		Preterm Birth Analysis Sample	
Term Live Births	2379	All Live Births except Term LBW	2511

Source of Bias in Prenatal Care Utilization Indices: Implications for Evaluating the Medicaid Expansion

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ABSTRACT

Background: Recent expansions in eligibility for coverage of prenatal care services by the Medicaid program reflect national initiatives to improve pregnancy outcomes. This study investigates the potential impact that completeness of reporting of prenatal care and gestational age variables and strategies to impute missing data may have on evaluations of the Medicaid expansion.

Methods: This study, examining 15 years of vital record data from a single state and comparing 1 year of data from four mid-Atlantic states, selected single live births to resident mothers for analyses. The "day 15" and the "preceding case" methods were used to impute missing gestational age data.

Results: Considerable temporal and geographic variation was detected in completeness of reporting of variables used to construct prenatal care indices. After imputing values for cases with missing data, the proportion of cases for which adequacy of prenatal care utilization could not be determined ranged from 3% to 24% among the states investigated. For those cases where gestational age data could be imputed, the distribution of prenatal care utilization was not markedly disparate from those cases with complete reporting of gestational age.

Conclusions: The results indicate that variations in reporting, decisions regarding the treatment of missing data, and the choice of the denominator can alter prenatal care utilization percentages and have implications for evaluations of the impact of the recent Medicaid expansion on prenatal care utilization. (*Am J Public Health.* 1991;81:1013–1016)

Introduction

Recent expansions in eligibility for coverage of prenatal care services by the Medicaid program reflect policy initiatives to meet Year 2000 national health objectives related to low birthweight and infant mortality.[1] Increased utilization of prenatal care has been associated with improved pregnancy outcomes.[2–7] In an effort to reduce financial barriers to prenatal care, Medicaid eligibility criteria were raised in 1990 to 133% of the federal poverty level, with state options to extend coverage to 185% of poverty. One focus for evaluation of this policy is the extent to which changes in eligibility for state Medicaid coverage will increase utilization of prenatal care and ultimately decrease low birthweight percentages and infant mortality rates.[8]

Prenatal care utilization is commonly measured by composite indices, the most notable being the Kessner Index of Adequacy of Prenatal Care.[2] This index considers the month prenatal care began, the number of prenatal care visits received, and the gestational age at delivery to categorize prenatal care utilization as adequate, intermediate, or inadequate. The adequate category includes cases with a first trimester initiation of care and prenatal care visits that are more than or equal to a recommended number for a pregnancy of a given duration. More visits are required as gestation increases. Initiation of care in the second trimester or an earlier initiation with less than the number of visits recommended to achieve the adequate category is termed intermediate care. Cases with no prenatal care, initiation of care in the third trimester, or an earlier initiation of care with less than the number of visits stipulated to achieve the intermediate category are labeled inadequate care. Modifications and alternatives to this index have been proposed[3,9–11] and used to investigate the relationship of prenatal care to a variety of pregnancy outcome measures.[2–6,9] Unfortunately, descriptions of these indices have often failed to specify how missing or implausible data should be handled.

The treatment of missing data in the construction of these indices is an important issue. In the Kessner index, cases with missing prenatal care data are classified as having inadequate prenatal care utilization.[2] Further, while various methodologies have been suggested to impute missing gestational age data,[12–15] a convention for the use of imputed gestational age data in the calculation of prenatal care utilization indices has not been established. The date of last menstrual period (LMP), needed to calculate the gestational age interval, has been estimated to be incomplete on approximately 20% of the nation's birth certificates.[12]

State-specific analyses of trends in prenatal care utilization before and after adoption of Medicaid options and comparisons of prenatal care among states with varying eligibility levels may be sub-

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This paper was submitted to the journal September 27, 1990, and accepted with revisions March 6, 1991.

TABLE 1—Percentage of Missing Components of Prenatal Care Utilization Index by 5-Year Groups: 1974–1988 South Carolina Resident Single Live Births

Missing Component	1974–1978	1979–1983	1984–1988
		Percent	
Trimester that prenatal care began[a]	5.99	0.74	1.10
No. of prenatal care visits[b]	1.09	0.62	1.04
Gestational age interval[c]			
No imputation	16.33	5.31	6.91
Day 15	2.09	1.97	2.21
Preceding case	2.04	1.92	2.15
Total missing[d]			
No imputation	19.05	5.37	7.03
Day 15	7.50	2.21	2.63
Preceding case	7.47	2.18	2.58

[a] Includes cases with a not-stated month prenatal care began or with a stated but nonspecific response, e.g., an actual month (January through December).

[b] Includes cases with a not-stated number of visits or with stated visits of 50 or more.

[c] Includes cases with an incomplete date of last menstrual period or a missing gestational age interval value after imputation, where indicated. Gestational age intervals <20 or >50 weeks are treated as missing.

[d] Includes cases with a missing value for any of the prenatal care utilization index component variables, i.e., trimester prenatal care began, number of prenatal care visits, and gestational age interval from last menstrual period.

TABLE 2—Prenatal Care Utilization Percentages by Gestational Age Imputation Method and 5-Year Groups: 1974–1988 South Carolina Resident Single Live Births

Prenatal Care Groups	1974–1978 (N = 237 347)	1979–1983 (N = 251 247)	1984–1988 (N = 256 175)
No imputation			
Adequate	48.36	55.81	53.61
Intermediate	24.43	29.23	28.45
Inadequate/missing	27.21	14.97	17.94
Missing[a]	19.05	5.37	7.03
Inadequate	8.17	9.59	10.91
Day 15			
Adequate	54.27	57.42	55.88
Intermediate	28.46	30.34	29.96
Inadequate/missing	17.27	12.23	14.16
Missing[a]	7.50	2.21	2.63
Inadequate	9.77	10.02	11.53
Preceding case			
Adequate	54.27	57.43	55.89
Intermediate	28.48	30.36	29.98
Inadequate/missing	17.25	12.21	14.13
Missing[a]	7.47	2.18	2.58
Inadequate	−9.78	−10.03	−11.55

[a] Includes cases with a missing value for any of the prenatal care utilization index component variables, i.e., trimester prenatal care began, number of prenatal care visits, and gestational age (after imputation where indicated).

Note: Because of rounding, columns may not total 100%.

ject to bias due to changes in the completeness of reporting of the variables used to construct these indices and in methodologies employed to impute missing data. This study investigates the potential impact that completeness of reporting of prenatal care and gestational age variables and strategies to impute missing data may have on evaluations of the Medicaid expansion. Two study designs have been employed: (1) examining 15 years of data from a single state, and (2) comparing 1 year of data from four eastern states.

Data and Methods

Computerized South Carolina live birth files for the years 1974 to 1988 were used for the first component of this investigation. For the second component, additional data from three mid-Atlantic states with varying levels of completeness of reporting of gestational age were drawn from the Department of Health and Human Services (DHHS) Region III Perinatal Information Consortium's 1987 Live Birth File.[16] Single births to resident mothers were selected for both analyses.

Gestational age was calculated as the interval from the date of last normal menses to the date of birth and was truncated into completed weeks.[17] Two strategies were employed for imputing missing gestational age data.[15] The "day 15" method imputes a value of 15 for the day of last normal menses for those births for which this single piece of information prevents the calculation of the gestational age interval. The "preceding case" method imputes gestational age by inserting the calculated gestational age interval value of a preceding birth with a corresponding month of LMP, 500-g birthweight interval, and race of mother.

Results

Table 1 displays the proportion of South Carolina cases with missing prenatal care and gestational age information for three 5-year periods. Nearly 6% of the cases in the 1974 to 1978 period exhibited missing or nonspecific data on month or trimester of pregnancy that prenatal care began. Missing gestational age data were found for more than 16% of the cases in the 1974 to 1978 period, but dropped markedly in later periods. After imputation methods were employed to recapture cases with incomplete dates of LMP, gestational age was still missing for approximately 2% of the cases in each period.

Table 2 presents percentages of prenatal care utilization employing three different treatments of missing gestational age data (no imputation, day 15, and preceding case). Based on no imputation, adequate prenatal care utilization was lowest in the 1974 to 1978 period (48.36%). When cases with missing data were combined with the inadequate category, the percentages based on no imputation varied across the three periods from 27.21% to 14.97% to 17.94%. The separate inadequate category increased from 8.17% in the 1974 to 1978 period to 10.91% for 1984 to 1988. There is little appreciable difference in the prenatal care distribution percentages produced by the two alternative methods for imputing missing gestational age values.

The next phase of the analysis utilized 1987 data from four states. The preceding case method was selected to impute missing gestational age values. Table 3 depicts by state the percentages of cases with missing gestational age data, before and after imputation, and missing prenatal care initiation and visit data. States B and C revealed a relatively higher proportion of missing gestational age data, 20.10% and 17.58%, respectively. While imputation markedly reduced this proportion in state C (5.73%), it had little impact in state B, where most cases with missing gestational age data were missing the entire date of LMP, not just the day value.

Prenatal care utilization percentages by state and gestational age imputation group are displayed in Table 4. These percentages are calculated in two different ways. In part A, percentages are based on a denominator of all live births with a separate category for cases with missing prenatal care or gestational age values after the application of the indicated gestational age imputation method. In part B, after imputation where indicated, cases with missing prenatal care and gestational age data are excluded, and percentages are based on a denominator of cases with the prerequisite in-range data elements needed to compute the index.

For states A and B, the imputation of gestational age had little appreciable effect on prenatal care percentages based on all cases (Table 4, part A). However, the proportion of total missing cases was noticeably reduced by imputing gestational age in the other two states. When calculations only included cases with in-range or imputed data values (Table 4, part B), the use of the gestational age imputation method had little appreciable impact on the prenatal care utilization percentages. This result suggests that the prenatal care utilization distribution for those cases for which a missing gestational age value could be imputed was only slightly less adequate than that of cases with complete gestational age data. For example, in state C, where gestational age was imputed for more than 10% of the cases, the adequate percentage was 71.55% before imputation and 71.06% afterward.

TABLE 3—Percentage of Missing Components of Prenatal Care Utilization Index by State: 1987 Resident Single Live Births

Missing Component	State A	State B	State C	SC[a]
Trimester that prenatal care began[b]	4.96	4.73	2.84	0.88
No. of prenatal care visits[c]	2.12	6.68	2.94	1.17
Gestational age interval[d]				
No imputation	5.10	20.10	17.58	7.26
Preceding case	4.69	20.01	5.73	2.16

[a] SC = South Carolina

[b] Missing trimester in pregnancy prenatal care began category includes cases with a not-stated month prenatal care began.

[c] Missing number of prenatal care visits category includes cases with a not-stated number of visits or with stated visits of 50 or greater.

[d] Missing gestational age category includes cases with an incomplete date of last menstrual period or a missing gestational age interval value after imputation, where indicated. Gestational age intervals of <20 or >50 weeks are treated as missing.

TABLE 4—Prenatal Care Utilization Percentages by Imputation Method and State: 1987 Resident Single Live Births

	State A	State B	State C	SC[a]
Part A: Percentages Based on All Cases				
No imputation	9929	70 835	158 468	51 580
Adequate	46.40	57.17	58.52	52.49
Intermediate	32.51	14.71	17.23	28.71
Inadequate	14.57	3.99	6.03	11.50
Missing[a]	6.52	24.13	18.21	7.30
Preceding case	9929	70 835	158 468	51 580
Adequate	46.57	57.22	65.90	55.02
Intermediate	32.68	14.73	19.87	30.29
Inadequate	14.60	3.99	6.97	12.14
Missing[a]	6.14	24.06	7.25	2.55
Part B: Percentages Based on Cases with a Calculated Prenatal Care Index Value (Missing Excluded)				
No imputation	9282	53 740	129 817	47 813
Adequate	49.63	75.36	71.55	56.62
Intermediate	34.78	19.39	21.06	30.97
Inadequate	15.59	5.25	7.39	12.41
Preceding case	9319	53 894	146 974	50 263
Adequate	49.62	75.34	71.06	56.46
Intermediate	34.82	19.40	21.42	31.08
Inadequate	15.56	5.26	7.52	12.46

[a] SC = South Carolina

[b] Includes cases with a missing value for trimester prenatal care began, number of prenatal care visits, or gestational age (after imputation where indicated).

Note: Because of rounding, columns may not total 100%.

Discussion

These data indicate that variations in reporting, decisions regarding the treatment of missing data, and choice of denominator can alter prenatal care utilization percentages. The true potential impact of the Medicaid expansion on prenatal care utilization may be obscured due to these factors. Therefore, evaluations of the Medicaid expansion should include a thorough examination of reporting completeness, a disclosure of strategies employed for imputing missing data, and a description of the methods used to calculate prenatal care percentages.

These data further suggest that it is unwise to categorize cases with missing prenatal care or gestational age data in the inadequate care group. The distribution of prenatal care utilization was not markedly disparate for those cases with imputed gestational age data and those cases with complete reporting of gestational age. This result argues both for employing gestational age imputation strategies and not assuming that cases with missing data had inadequate prenatal care utilization. While the prenatal care utilization of those cases with missing data remains unknown, estimates of inadequate prenatal care usage that include cases with missing data may be inflated.

Whether prenatal care percentages should be calculated from all cases or only those for which the index can be generated is illustrated by data from state B. Based on all cases, 57% of women delivering in that state were placed in the adequate category. Excluding cases with missing data from the calculation resulted in a placement of 75% of women into the adequate category. The ease with which prenatal care percentages can be manipulated into adequate and other than adequate categories should not be ignored, given the political context of an impending evaluation of a major public policy initiative. If we assume that cases for which the index can be calculated are roughly similar or only marginally better in their prenatal care utilization patterns than those without the prerequisite data, the 75% figure may be a better estimate of the proportion of all births in the state with adequate prenatal care utilization. To alternatively assume that most cases with missing data have less than adequate utilization of prenatal care is unrealistic in areas with large proportions of missing data. While cases with missing gestational age data have been shown to display higher risks of poor pregnancy outcomes,[15] all cases with missing data will not necessarily exhibit adverse outcomes or less than adequate prenatal care utilization. Care should be taken in the analysis and reporting of these percentages to ensure that large missing group percentages are not used to represent individuals with less than adequate prenatal care utilization.

The choice between the day 15 or the preceding case method to impute missing gestational age data appears to have little impact on prenatal care utilization distributions. While the day 15 method has been noted to inflate preterm percentages in comparison with the preceding case method,[12,15] the amount of bias introduced by this approach appears insufficient to produce a prenatal care utilization distribution markedly different from that obtained by the preceding case method.

Prenatal care utilization indices are a useful but crude surveillance tool for assessing needs and evaluating programs and policy. Evaluation of the Medicaid expansion by these indices is a heuristic attempt to provide accountability for a major policy initiative that may enhance maternal and infant health in ways not easily appraised by available health measurement tools. Because the components of these indices are subject to reporting bias, and because important characteristics of prenatal care (e.g., quality, comprehensiveness, and continuity) are not considered, it is imperative that these indices be prudently employed in evaluation efforts and be continually scrutinized for bias.

Credible data for programmatic efforts and policy discourse require both accurate and complete reporting and logical and systematic analysis. The three component variables of prenatal care utilization indices contain potentially large measurement error. Vagaries in analytical methods used to compute these indices can further add to the uncertainty regarding the proposed relation between prenatal care utilization and pregnancy outcome and the possible impact of policy initiatives. These methodological concerns require ongoing attention, but they should not be used to divert attention from critically needed efforts to ensure that all women have access to and are encouraged to use prenatal care. □

Acknowledgment

This manuscript was presented in part at the 1990 annual meeting of the American Public Health Association in New York City.

References

1. From the Health Resources and Services Administration. Office of Maternal and Child Health. *JAMA.* 1989;261:199. Editorial.
2. Kessner DM, Singer J, Kalk CW, et al. Infant death: an analysis of maternal risk and health care. *In: Contrasts in Health Status*, I. Washington, DC: Institute of Medicine, National Academy of Sciences, 1973.
3. Gortmaker SL. The effects of prenatal care upon the health of the newborn. *Am J Public Health.* 1979;69:653–660.
4. Showstack JA, Budetti PO, Minkler D. Factors associated with birthweight: an exploration of the roles of prenatal care and length of gestation. *Am J Public Health.* 1984;74:1003–1008.
5. Alexander GR, Cornely DA. Racial disparities in pregnancy outcomes: the role of prenatal care utilization and maternal risk factors. *Am J Prev Med.* 1987;3:254–261.
6. Murray JL, Bernfield M. The differential effect of prenatal care on the incidence of low birth weight among whites and blacks in a prepaid health care plan. *N Engl J Med* 1988;319:1385–1391.
7. Institute of Medicine. *Preventing Low Birth Weight.* Washington, DC: National Academy Press, 1985.
8. Hogue C. Conference opening remarks presented at the Medicaid Expansion and Infant Mortality Workshop; June 1989; Bethesda, Md.
9. Alexander GR, Cornely DA. Prenatal care utilization: its measurement and relationship to pregnancy outcome. *Am J Prev Med.* 1987;3:243–253.
10. McDonald TP, Coburn AF. Predictors of prenatal care utilization. *Soc Sci Med.* 1988;27:167–172.
11. Kotelchuck M. The mis-measurement of prenatal care adequacy in the US and a proposed alternative two-part index. Presented at the APHA Annual Meeting; October 1987; New Orleans, La.
12. Taffel S, Johnson D, Heiser R. A method of imputing length of gestation on birth certificates. *Vital Health Stat.* [2]. 1982; No. 92.
13. Williams RL, Creasy RK, Cunningham GC, et al. Fetal growth and perinatal variability in California. *Obstet Gynecol.* 1982;59:624–632.
14. David RJ. Population-based intrauterine growth curves from computerized birth certificates. *South Med J.* 1983;76:1401–1406.
15. Alexander GR, Tompkins ME, Cornely DA. Gestational age reporting and preterm delivery. *Public Health Rep.* 1990;105:267–275.
16. Zinzeleta E: DHHS region III perinatal information consortium. Baltimore, Md: Johns Hopkins University; 1989. Technical Report 89-1.
17. Silverman WA, Lucey JF, Beard A, et al. Nomenclature for duration of pregnancy, birth weight and intrauterine growth. *Pediatrics.* 1967;39:935–939.

Uncompensated Hospital Care for Pregnancy and Childbirth Cases

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ABSTRACT

Background: The large number of medically indigent patients in the United States is a major concern to policymakers and may be due to recent increases in the number of uninsured people. The purpose of this study was to identify the factors that affect the amount of unpaid hospital charges for services provided to pregnant women.

Methods: Individual and hospital data were collected on a representative set of 235 pregnancy and childbirth patients with unpaid hospital charges from 28 hospitals in the state of Indiana.

Results: Most of these patients did not have insurance coverage (63.8%), yet the majority were employed in the public or private sector (72.3%). Over half (55.5%) of the total uncompensated care amount for this group was from the $1000 to 2499 debt category. The median charge for these patients was $1468, of which the typical hospital was able to collect only 25.5%.

Conclusions: The findings support the belief that any national effort to expand the availability of health insurance coverage to women through increased employment will not totally eliminate the uncompensated care problem. The findings also indicate that rural hospitals face the uncompensated care problem mainly because a significant portion of rural patients are without adequate health insurance coverage. (*Am J Public Health.* 1991;81:1017–1022)

Introduction

The large number of medically indigent patients in the United States, a major concern to policymakers, can be attributed to increases in the number of uninsured and underinsured people. This increase in indigent patients, especially pregnant women, has created additional financial burdens for hospitals. In our earlier studies examining uncompensated care patients in hospitals,[1,2] we found that pregnancy and childbirth accounted for the largest proportion of uncompensated care cases (19.1%) and the largest percentage of uncompensated dollar amounts (17.4%). Researchers have found that a lack of adequate prenatal care leads to low birthweight in infants, which in turn contributes to a higher incidence of infant mortality.[3–7] Other research indicates that younger women who have lower incomes and less education, reside in rural areas, or are Medicaid recipients tend to have less access to prenatal care.[3,6,8–11] Until recently, the eligibility requirements for Medicaid had been increasing, and as a result, relatively fewer women and children were being covered by Medicaid.[3,12–15] Denied access to prenatal care increases the hospital cost because of higher frequency of subsequent complications in delivery and perinatal care, which leads to even higher amounts of unpaid hospital bills.[4,7]

In this study, patients belonging to the pregnancy and childbirth subset of the original sample of uncompensated care inpatients[1] were examined to identify the factors that affect the amount of unpaid hospital charges for services provided to pregnant women. The identification of these factors could lead to the development of policies designed to remove existing patient, provider, and system barriers to prenatal care[3,4–6,16–18] and facilitate medical care access for pregnant women.

Methods

Uncompensated care normally consists of bad debt, charity care, contractual allowances, and courtesy discounts. The uncompensated hospital care costs considered in this study were only those originating from the bad debt and charity care categories. Medicare and Medicaid patients were also excluded because bad debts arising from their failure to pay deductibles are reimbursed by the Medicare and Medicaid programs. Bad debts are amounts due from patients who are expected to pay, but for some reason do not. Charity care is care that is provided to patients who, prior to admission, are generally identified as unable to pay and who are not expected to pay. The hospitals in this study did not consistently classify patients as "bad debt" or "charity care" cases.

The sampling scheme used in this study selected a representative set of Indiana hospitals as well as a representative set of patients from those hospitals with unpaid hospital charges. First, a stratified

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This article was submitted to the journal August 6, 1990, and accepted with revisions April 23, 1991.

TABLE 1—Total Number and Percentage of Uncompensated Care Pregnancy/Childbirth Cases by Dollar Category

	N	Total Sample, %	0–$499, %	$500–$999, %	$1000–$2499, %	$2500–$4999, %	$5000–$9999, %	χ^2 *P* Value
Total uncompensated care cases	235	100.0	22.6	15.7	50.2	10.4	1.3	
Location								
Urban	150	63.8	69.8	59.5	62.7	62.5	66.7	.875
Rural	85	36.2	30.2	40.5	37.3	37.5	33.3	
Hospital size								
Large	98	41.7	49.1	43.2	37.3	41.7	66.7	.611
Medium	39	16.6	17.0	10.8	20.3	8.3	0.0	
Small	98	41.7	34.0	45.9	42.4	50.0	33.3	
% Originating from hospital's county	174	79.1	78.0	81.8	80.7	66.7	100.0	.583

random sample based on urban/rural classification of 27 of the 115 acute care general hospitals in Indiana was drawn. Additionally, a tertiary teaching hospital was purposely included in the study to represent that class of hospital. Of the 28 hospitals, 14 (50%) were classified as urban, 16 (57.1%) as small (fewer than 150 beds), 5 (17.9%) as medium (150 to 399 beds), and 7 (25%) as large. All large hospitals were located in urban areas. Second, once the sample of hospitals was identified, a systematic random sample of those patients who did not pay all of their hospital bill during the 1986 calendar year was selected by using a 1/20 sampling fraction. This sampling scheme identified 235 (13.9%) patients hospitalized with the primary diagnosis of pregnancy and childbirth who received uncompensated care. Total revenue for the study hospitals was compiled from the *1986 Indiana State Board of Health Financial Disclosure Report.*[19]

The pregnancy and childbirth cases receiving uncompensated hospital care in our sample were classified by urban/rural hospital location on the basis of whether the hospital was located within a Standard Metropolitan Statistical Area and by amount of the unpaid hospital bill of the patient.

The data were analyzed with the Student's *t* test and analysis of variance to test for significant differences in means; tests for independence were assessed with the chi-square test. The criterion for statistical significance was $P < .05$.

A multiple regression model was constructed by using the percentage of the charges left unpaid as the dependent variable. Potential explanatory variables were included in a stepwise-forward procedure (using $P < 0.10$ for inclusion) to determine which variables had a significant impact on the dependent variable, controlling for the other variables. Since the interval-ratio scale variables used in the model were skewed, log transformed versions of these variables were used in the model to improve their normality.

The results of this study should be interpreted cautiously because of the inherent limitations of the study data. This study was partly based on secondary patient data reported to the hospital by individual patients upon hospital admission. Also, the employment data did not distinguish between part-time and full-time employment, they merely indicated whether a person was reported as being employed or not.

Results

As shown in Table 1, 61.9% of the 235 cases had unpaid hospital charges of $1000 or more. Fewer than one fourth of the cases (22.6%) left unpaid charges under $500. Nearly two thirds (63.8%) were treated in urban hospitals with 41.7% treated in large hospitals, another 41.7% in small hospitals, and only 16.6% treated in medium-sized facilities. On average, 79.1% of the pregnancy and childbirth patients with unpaid bills originated from the county in which the hospital was located. There were no statistically signincant relationships between the distribution of the unpaid amount and the location of the hospital, the size of the hospital, and the number of uncompensated care patients originating from the same county as the hospital.

The study group received a total of $311 382 in uncompensated care (Table 2). Individual uncompensated care amounts ranged from $4 to $7975. Less than 5% of the total uncompensated care amount was from debts smaller than $499; over half (55.5%) of the total uncompensated care amount was from the $1000 to 2499 debt category. Patients in this study with unpaid hospital charges of $5000 or more were responsible for only 6.4% of the uncompensated care.

The total hospital charges for these patients was $469 999, of which study hospitals were only able to collect an average 25.5%. The median total hospital charges for these cases was $1468 with a median uncollected amount of $1226 (83.5%). Patient characteristics, as summarized in Table 3, reveal that the typical pregnancy and childbirth patient with unpaid hospital charges was 23.3 years old, was married (61.8%), spent 3.2 days in the hospital, and was discharged to self/home care (84.7%). The age and marital status of the patients showed no significant relationships with the amount of the hospital bill left unpaid. As the length of stay increased, so did the amount of the hospital bill left unpaid. In addition, a larger proportion of the patients with greater amounts of unpaid hospital bills were discharged to long-term health care facilities. Those with insurance coverage tended to have fewer unpaid hospital charges, and those without insurance tended to leave larger amounts unpaid.

The majority (77.6%) of the study patients reported to be employed in either the public or private sector or self-employed, yet only 36.2% had some form of health insurance coverage. The unemployed group left significantly higher amounts of their hospital bill unpaid than the patients who were employed.

A second analysis evaluated patient and hospital variables by dollar amount of uncompensated care as shown in Table 4. Nearly two thirds (63.2%) of the uncompensated amount was reported by urban hospitals. However, the mean uncompensated care dollar amounts per case did not

TABLE 2—Selected Financial Characteristics of Uncompensated Care Pregnancy/Childbirth Patients

	Total Sample	Less than $500	$500–$999	$1000–$2499	$2500–$4999	$5000–or More
Total uncompensated care (% of total)	$311 382	$14 901 (4.8%)	$28 439 (9.1%)	$172 688 (55.5%)	$75 401 (24.2%)	$19 953 (6.4%)
Total charged (% of total)	$469 999	$87 003 (18.5%)	$52 381 (11.1%)	$193 430 (41.1%)	$117 232 (24.5%)	$19 953 (4.2%)
Average percentage of charges that is collected	25.5%	76.5%	25.3%	6.9%	7.9%	0.0%
Median total charge to uncompensated care patients	$1468	$1286	$961	$1480	$3211	$6535
Median uncollected amount	$1226	$297	$800	$1365	$2970	$6535

TABLE 3—Uncompensated Care Pregnancy/Childbirth Patients by Selected Demographic Characteristics

	N	Total Sample, %	Less than $500, %	$500–$999, %	$1000–$2499, %	$2500–$4999, %	$5000 or More	*P* Value
Mean age (years)		23.3	23.7	22.4	23.4	23.6	22.0	.734[a]
Distribution of age, y								
0–17	22	9.4	9.4	8.1	9.3	12.5	0.0	.819[b]
18–39	213	90.6	90.6	91.9	90.7	87.5	100.0	
Marital status								
Married	126	61.8	67.3	66.7	56.9	66.7	50.0	
Single adult	76	37.3	32.7	33.3	41.2	33.3	50.0	.761[b]
Widowed	2	1.0	0.0	0.0	2.0	0.0	0.0	
Average length of stay (days)		3.2	3.0	2.5	3.0	4.7	9.3	.000[a]
Discharge status								
Self/home care	199	84.7	92.5	83.8	86.4	66.7	33.3	
SNF/ICF	35	14.9	7.5	16.2	12.7	33.3	66.7	.004[b]
Dead	1	0.4	0.0	0.0	0.8	0.0	0.0	
Primary insurance								
Self-pay	150	63.8	17.0	75.7	83.1	58.3	33.3	.000[b]
Commercial and Blue Cross	63	26.4	71.7	16.2	10.2	25.0	0.0	
Other	23	9.8	11.3	8.1	6.8	16.7	66.7	
Employment status								
Unemployed	42	22.3	6.7	15.6	31.2	27.8	33.3	
Public and private sector	136	72.3	93.3	75.0	61.1	72.2	66.7	.0150[b]
Self-employed	10	5.3	0.0	9.4	7.8	0.0	0.0	

Note: Because of rounding, some percentage numbers do not add up to 100.

[a] *t* test for mean values.

[b] χ^2 test.

differ significantly for the rural and urban hospitals. Large hospitals incurred 40.8% of the uncompensated care dollars; medium-sized hospitals, 16.1%; and small hospitals, 43.1%. There was no significant difference in the mean uncollected amounts between the three classes of hospital size.

Self-pay patients accounted for 71.0% of the dollar amount of unpaid hospital bills, compared with 16.4% for commercial and Blue Cross patients. Self-pay patients also accounted for significantly larger mean uncollected amount (along with the "other insurance" category). Of the total uncompensated amount, only 26.5% was generated by those who were unemployed while over two thirds (68.7%) was generated by people employed in the private or public sector. However, there was not a significant difference between the employment groups in the mean amounts of uncompensated care. Married patients accounted for 61.3% of the uncompensated dollar amount, but the mean uncompensated amounts did not differ significantly between the marital status groups.

Table 5 presents uncompensated care information by hospital location. Of the cases treated in urban hospitals, most (65.3%) were in large facilities. Slightly more urban than rural hospital patients resided in the county the hospital was located in, but the differences were not statistically significant.

The study subjects from the urban and rural hospitals did not differ significantly with respect to age, marital status, average length of stay, or employment status. However, cases with unpaid hospital charges reported from rural hospitals were more likely to be self-insured (74.1% vs 58.1%) and more likely to be discharged to self/home care (95.3% vs 78.7%). The median charge for pregnancy and childbirth care for the group who did not pay all of their hospital charges was somewhat higher for those treated in urban hospitals. However, the mean uncollected amount did not differ between the urban and rural hospitals because of the higher collection rates of the urban hospitals.

The multiple regression procedure indicated that two insurance categories: "Commercial and Blue Cross" and "Other" (i.e., HMO, PPO) contributed the

TABLE 4—Amount and Percentage of Total Uncompensated Care Dollars for Pregnancy/Childbirth Patients

	Amount of Uncompensated Care	Percentage of Total	Mean Amount of Uncompensated Care	ANOVA *P* Value
Location				.815
Urban	$196 924	63.2	$1313	
Rural	$114 458	36.8	$1346	
Hospital size				.866
Large	$127 001	40.8	$1296	
Medium	$50 232	16.1	$1288	
Small	$134 149	43.1	$1369	
Primary insurance				.000
Self-pay	$221 111	71.0	$1474	
Commercial and Blue Cross	$50 920	16.4	$821	
Other	$39 351	12.6	$1711	
Employment status				.266
Unemployed	$64 726	26.5	$1541	
Private and public sector	$168 131	68.7	$1235	
Self-employed	$11 759	4.8	$1176	
Marital status				.972
Married	$162 007	61.3	$1266	
Single	$99 341	37.6	$1307	
Widowed	$2 853	1.1	$1426	

TABLE 5—Selected Patient and Hospital Characteristics by Location and Hospital Size for Pregnancy/Childbirth Patients

	Location		
	Urban	Rural	*P* Value
Uncompensated care cases, %	63.8	36.2	
Patients by hospital size, %			.000[a]
Large	65.3	0.0	
Medium	26.0	0.0	
Small	8.7	100.0	
Originating from hospital county, %	81.5	75.3	.272[a]
Average age, y	23.0	23.8	.257[b]
Marital status			.671[a]
Married	61.1	63.6	
Single adults	37.6	36.4	
Widowed	1.3	0.0	
Primary insurance			.0153[a]
Self-pay	58.0	74.1	
Commercial and Blue Cross	28.7	22.4	
Other	13.3	3.6	
Average length of stay (days)	3.3	3.0	.405[b]
Discharge status, %			.000[a]
Self/home care	78.7	95.3	
SNF/ICF	21.3	3.5	
Dead	0.0	1.2	
Employed, %	64.7	74.1	.135[a]
Total amount of uncompensated care	$196 924	$114 458	
Total amount charged	$326 231	$143 768	
Average percentage of charged amount that is collected	29.7	18.1	.016[b]
Median total charge to uncompensated care patients	$1555	$1276	.059[b]
Median uncollected amount	$1188	$1230	.815[b]

[a] χ^2.

[b] *t* test on mean values.

greatest amount to the model. The amount of the total charge to the patient was also significant at the $P < .10$ level. These three variables accounted for nearly 40% of the variation in the percentage of the hospital charges left unpaid. For all three variables, the partial regression coefficients were negative; this indicated that if the patient had some form of insurance, the percentage of the bill left unpaid was lower, and if the total charge was higher, the percentage left unpaid was also lower. Demographic characteristics of the patients (age, marital status, employment) were not found to be important factors in the model. Neither were the discharge status and the length of stay. The two hospital characteristics, urban/rural status and total revenue, were also not important factors in the model.

Discussion

The study of pregnancy and childbirth patients who do not pay all of their hospital bill resulted in three findings that suggest a need to further examine existing public policy as it affects these women: (1) the majority (63.8%) of the pregnancy and childbirth patients who left part or all of their hospital bill unpaid did not have insurance; (2) pregnancy and childbirth patients tended to have overall larger dollar amounts of bad debts relative to other uncompensated care patients; and (3) the collection experience was lower for pregnancy and childbirth patients than for patients from all diagnosis groups as previously reported.[1,2]

The data indicate that the patient's insurance coverage was the major factor in a facility's ability to recover its total charges. Only a third of the patients in this study had any insurance coverage. It is remarkable that 63.8% of the pregnancy and childbirth patients with unpaid hospital charges had no insurance coverage as compared with 44.6% who had no insurance in the sample of patients from all diagnosis groups found in our earlier studies.[1,2] This is a high proportion of uninsured patients in light of the fact that over three fourths of the pregnancy and childbirth patients were reportedly employed. There are three possible explanations: (1) some of the employers do not offer health insurance benefits to their employees, (2) the individual cannot afford or for other reasons chooses not to purchase the insurance, (3) or the insurance policies do not cover pregnancy and childbirth-related hospital services (there may be a waiting period for maternal benefits to take effect). Some of the insurance plans may have high coinsurance or deductibles for pregnancy-related health care, and those patients with insurance coverage may be unable or unwilling to pay their portion of the hospital bill.

These findings, although based solely on the findings of one state, may support the belief that any national effort to expand the availability of health insurance coverage to women through employers will not totally eliminate the uncompensated care problem for maternal and child health patients and the hospitals providing the care. The fact that the majority of the women in this group were employed (77.6%) points to a different set of problems. A policy expanding availability should be considered a step in the right direction to reduce the amount of uncompensated care provided by hospitals. Yet the greater problem may be that employed women in this group cannot afford the insurance premiums and thus remain uninsured or underinsured. On the other hand, if high coinsurance amounts and large deductibles are problems for those with insurance (36.2%), changes in the shared portion would also be required to reduce the amount of the hospital charges determined to be the responsibility of the patients themselves (i.e., lower copayment or deductible amounts).

A second major contrast between the results found in this study of pregnancy and childbirth patients and our previous study of all patients was that pregnancy and childbirth patients tended to have larger total uncompensated dollar amounts. Fewer than 5% of the pregnancy and childbirth cases had uncompensated care amounts of less than $500 compared with 39.2% in the sample of patients from all diagnosis groups. Over one half of the pregnancy and childbirth cases had uncompensated care amounts between $1000 and $2499, compared with only 29.2% in the overall study. This clearly indicates that pregnancy and childbirth patients represent a serious financial problem to the hospital.

The third finding shows that of the total charges to pregnancy and childbirth patients, only a small portion was eventually collected (25.2% compared with 38.8% overall). This points to an apparent inability and/or unwillingness of the facility to seek recovery of these outstanding balances. Hospital administrators may perceive that aggressively pursuing these unpaid bills may be futile and/or not cost-effective on an individual basis, even though collectively such cases represent a large proportion of the hospital's total uncompensated care. The indirect cost of collecting these debts, in the form of poor publicity generated in the community, may be seen as somewhat counterproductive to the mission of the facility.

Although the median dollar amount charged by rural facilities was lower than that charged by urban facilities, the collection rate was also lower, thus the median uncollected amount was approximately the same. These findings indicate that rural hospitals face the uncompensated care problem mainly because a significant portion of rural patients are without adequate health insurance coverage. If this is the case, there is a need to expand insurance coverage to the rural population to ensure that this population has access to comprehensive health care.

The patterns seen with uncompensated hospital care may be related to the overall problem of inadequate prenatal care. Some of the patients who leave hospital bills unpaid may also have failed to obtain adequate prenatal care. The major factor thought common to both problems involves financial barriers to care. Thus hospital administrators are faced with the difficult question of how aggressively they should seek payment for services rendered. Aggressive collection efforts may discourage the medically indigent, higher risk women from seeking prenatal care. However, if adequate prenatal care is not provided by the hospital (even at a loss), high-risk women are more likely to have medical complications. Thus, when these women are finally admitted into the facility, the result could be an even greater amount of uncompensated care than if they had received adequate prenatal care initially. Evidence shows that better prenatal care would be obtained if the financial barriers for patients were removed.[3,6,8–11]

Increasing health insurance coverage for the patients, and not just increasing the availability of health insurance for both those employed and unemployed, is a possible solution. However, an increase in insurance coverage entails more costly public policy interventions. One public policy option could include requiring mandatory insurance coverage to be provided (not merely made available) by employers. However, this option may result in higher cost of goods and services, making American businesses even less competitive in the international arena than they already are. A second option would be to increase Medicaid coverage and eligibility. This option would result in higher taxes and/or the shifting of resources from other public programs into health care programs. These possible consequences must be considered before any policy is enacted. However, we need to recognize that presently a large part of the burden of providing prenatal and hospital care to the indigent population falls on hospitals and only more indirectly on business or the taxpayer. If inroads are to be made into solving the infant mortality problems, policies regarding equitable compensation to hospitals must also be addressed. Further research is needed to examine the demographics and collection experiences of all hospital patients so that the characteristics of the paying and the nonpaying patients can be distinguished. ☐

Acknowledgments

This paper was presented at 118th Annual Meeting of the American Public Health Association on October 4, 1990, in New York City.

The authors would like to acknowledge the financial support of Blue Cross-Blue Shield of Indiana, the Indiana Hospital Association, the Lincoln National Corporation, and the Indiana University School of Public and Environmental Affairs.

References

1. Saywell RM, Zollinger TW, Chu DKW, MacBeth CA, Sechrist ME. Hospital and patient characteristics of uncompensated hospital care: policy implications. *J Health Polit Policy Law.* Summer 1989;14:287–307.
2. Zollinger TW, Saywell RM, Chu DKW, Ziegert A, Woods JR, LaBov D. A determination of institutional and patient factors impacting uncompensated hospital care. *Hosp Health Serv Adm.* Summer 1991;36:243–255.
3. Braveman P, Oliva G, Miller MG, Schaaf VM, Reiter R. Women without health insurance: links between access, poverty, ethnicity and health. *West J Med.* 1988;149:708–711.
4. Carter ER. Quality maternity care for the medically indigent. *MCN.* March–April 1986;11:85–92.
5. Cagle CS. Access to prenatal care and prevention of low birth weight. *MCN.* July–August 1987;12:235–238.
6. McDonald TP, Coburn AF. Predictors of prenatal care utilization. *Soc Sci Med.* 1988;27:167–172.
7. Murray JL, Bernfield M. The differential effect of prenatal care on the incidence of low birth weight among blacks and whites in a prepaid health care plan. *N Engl J Med.* 1988;319:1385–1391.
8. Gold RB, Kenney AM. Paying for maternity care. *Fam Plann Perspect.* May–June 1985;17:103–111.
9. St. Clair PA, Smeriglio VL, Alexander CS, Celantano DD. Social network structure and prenatal care utilization. *Med Care.* 1989;27:823–831.
10. Smith VK. Data show lack of insurance a serious problem. *Mich Med.* April 1989;88:19.
11. Fisher ES, LoGerfo JP, Daling JR. Prenatal care and pregnancy outcomes during the recession: the Washington state experience. *Am J Public Health.* 1985;75:866–869.
12. Rosenbaum S, Hughes DC, Johnson K.

Maternal and child health services for medically indigent children and pregnant women. *Med Care.* 1988;26:315–332.
13. Blendon RJ, Aken LH, Freeman HE, Kirkman-Liff BL, Murphy JW. Uncompensated care by hospitals or public insurance for the poor: does it make a difference? *N Engl J Med.* 1986;314:1160–1163.
14. Shahoda T. Uncompensated care costs rise nationwide. *Hospitals* March 5, 1987;61:34.
15. Marshall WJ The uninsured: a lack of realpolitik. *Ohio Med.* August 1989;85:591–594.
16. Shearer M. Health care for the uninsured. *Mich. Med.* April 1989;88:14–16.
17. Brown SS. Drawing Women into Prenatal Care. *Fam Plann Perspect.* March–April 1989;21:73–80.
18. Hubbel FA, Waitzkin H, Rucker L, Akin BV, Heide MG. Financial barriers to medical care: a prospective study in a university affiliated community clinic. *Am J Med Sci.* 1989;297:158–162.
19. Indiana State Board of Health. *1986/87 Indiana State Board of Health Disclosure Report.* Indiana State Board of Health; 1988.

Report Cites Vaccine Shortage in Health Centers Serving Poor Children

In 1990, the year that there were to be no more than 500 cases of measles nationally under goals set in 1979 by the Surgeon General, more than 25 000 cases of measles were reported. As community health centers grappled with shortages of government-supplied vaccines, according to a new Children's Defense Fund report, *Vaccine Supply and Low Income Children: Barriers Faced by Community and Migrant Health Center Patients*, more children died from measles in 1990 than in any year since 1971.

"Measles is a particularly sensitive and early indicator of the nation's eroding childhood immunization system because the disease is so contagious," said Sara Rosenbaum, director of CDF's health division. "Other diseases such as mumps and pertussis are showing similar surges. The current epidemic of utterly preventable childhood illness and death must be halted. We have only our lack of collective national will to blame for this crisis."

Yet in the midst of the worst measles outbreak in more than 20 years, the CDF report finds that federally funded community health centers that provide primary health care to nearly 3 million poor children have been experiencing shortages of government-supplied vaccines critical to immunizing poor children against childhood diseases. As a result, the youngest health center patients, all of which live in medically underserved urban and rural communities, run a high risk of inadequate vaccination.

Rosenbaum recommended a number of steps to alleviate the crisis in vaccine shortages and immunization delivery. One step is the immediate expansion of the Centers for Disease Control's (CDC) vaccine contract purchasing program by $80 million.

Rosenbaum said: "Currently CDC simply does not have enough publicly purchased vaccines to meet the needs of both state and local health agencies and health centers. All of these clinics must be assured an adequate supply of vaccines at the lowest available price. The CDC purchase and distribution contract system must be expanded at least to meet the needs of all publicly funded health care providers rather than forcing those providers to stop vaccinations or to purchase on the private market at four times the cost."

Moreover, according to the report, numerous health centers reported that state Medicaid agencies were not reimbursing them for the cost of vaccines administered to Medicaid-enrolled children which they were forced to purchase privately when their health department supplies were exhausted. Others reported that some states refused to cover certain vaccines at all despite federal mandates.

Rosenbaum pointed out that the National Vaccine Advisory Committee in January concluded that low vaccination rates and disease outbreaks are chiefly the result of systemic breakdowns, not parents' willful or negligent failure to seek vaccinations.

The CDF report reviews a number of systemic problems that have led to the growing vaccine crisis, including soaring vaccine costs over the past decade, the increasing number of private physicians who no longer vaccinate children in their offices, increased childhood poverty, and growth in the number of children with no health insurance.

"The childhood immunization crisis is a stark reminder of the severe shortage of basic health services that persist in countless medically underserved urban and rural communities," Rosenbaum said.

If recent trends continue, by the end of the century US immunization rates for infants and toddlers will lag behind those of dozens of Third World countries. Among the 16 countries with higher immunization rates than ours in 1988 to 1989 (as measured by 1-year-olds fully immunized against polio) were Bulgaria, Hungary, Greece, Brazil, China, Mexico, North Korea, Chile, and Rumania. Among the 55 countries with infant polio immunization rates higher than the United States non-White rate were Albania, Cuba, Jordan, Tunisia, Colombia, Iran, Malawi, Botswana, Maritius, Sri Lanka, Mongolia, Jamaica, and Rwanda.

Sociodemographic Factors and the Quality of Prenatal Care

ABSTRACT

Background: In this study, maternal sociodemographic factors are examined in relationship to the quality of prenatal health services US women receive.

Methods: Data from the 1980 National Natality Survey and 1980 Fetal Mortality Survey were used for the analysis. Indicator variables for prenatal care quality are the percentages of prenatal visits at which blood pressure and urine were tested, the performance of hemoglobin or hematocrit tests, and the presence or absence of advice regarding salt restriction and diuretics usage during pregnancy.

Results: Distribution of the basic examinations in prenatal care vary according to marital status, parity, education, and residence in a metropolitan or nonmetropolitan county. The advice received concerning salt and diuretics usage was also influenced by sociodemographic variables.

Conclusions: The analyses reveal that prenatal care is not of even minimally acceptable quality for many women. (*Am J Public Health.* 1991;81:1023–1028)

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Introduction

Numerous efforts have been made to assess the adequacy of prenatal care obtained by pregnant women in the United States. The adequacy of prenatal care can be measured by examining two components: (1) the timing and quantity of visits and (2) the quality of health services. With little exception to date,[1] studies of the adequacy of prenatal care have concentrated on the first component. Researchers have found that about one third of pregnant women obtain too few prenatal visits or start prenatal care too late. The groups getting the most inadequate care are teenagers, minorities, the least educated, the unmarried, and the poor[2–5] (P. J. Placek, unpublished manuscript, May 15, 1987; a more extensive report on this topic was prepared for the Institute of Medicine by Paul Placek, PhD in his private capacity, with appropriate clearances, and no official support or endorsement by his employer is intended or inferred.) Based upon data from the 1980 National Natality Survey (NNS) and the 1980 National Fetal Mortality Survey (NFMS), this research adds measures of quality to the study of the adequacy of prenatal care in the United States.

Women have varying degrees of control over the timing and quantity of prenatal health services they obtain, because sociodemographic attributes and health beliefs affect their utilization patterns. (When asked about barriers to obtaining adequate prenatal care, 39% of the Medicaid recipients and uninsured women interviewed by the US General Accounting Office cited attitudinal barriers, e.g., not wanting to tell others, being afraid of the pregnancy, and being afraid of the medical tests.)[6] Once a woman has chosen her physician, control over the quality of her health care shifts almost completely to the provider. Theoretically, the woman's sociodemographic attributes should not affect the quality of the services received.

The specific research questions to be answered are whether prenatal care recipients obtain basic examinations recommended by the American College of Obstetrics and Gynecology (ACOG), whether women receive appropriate medical advice concerning the use of salt and diuretics during pregnancy, and whether variability in the quality measures (surveillance and advice) corresponds to maternal sociodemographic characteristics.

ACOG recommends that all pregnant women receive both blood pressure examinations and urine tests, for protein and sugar, at every visit and that hematocrit and hemoglobin levels be assessed at least once during prenatal care.[7] Actual practices are compared to these standards of care.

Although attitudes about salt use in pregnancy had changed over time, by 1977 the ACOG had set the standard that normal women should use salt at the level preferred.[7,8] In other words, physicians should not normally advise salt restriction. Administration of diuretics during pregnancy may be acceptable treatment of underlying hypertensive disease but is not approved for normal pregnancy or for preeclampsia.[9,10] The kinds of advice

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This paper was submitted to the journal May 15, 1990, and accepted with revisions April 4, 1991.

TABLE 1—Explanatory Variables Included in the Regression Models[a]

Variables	Explanation
Maternal sociodemographic factors	
Age	Number of years of age at delivery
Non-Hispanic White[b]	Non-Hispanic White racial-ethnic group
Black[b]	Black racial group
Native American[b]	Native American Indian racial-ethnic group
Asian/Pacific Islander[b]	Asian or Pacific Islander racial-ethnic group
Education	Number of years of education with final value of 17 or more years[c]
Graduate	17 or more years of education
Married	Married at delivery
Parity	Number parity at start of current pregnancy
Metropolitan	Lived in metropolitan county at time of delivery
Prenatal care (PC) quantity and timing measures[d]	
Month PC begun	Month of pregnancy that first prenatal visit made
Number visits	Number of prenatal visits with final value of 30 or more visits[e]
Top visits	30 or more visits made
Maternal health status factors[f]	
High BPs %	Percent of prenatal visits at which blood pressures were above 140/90
Any complications	One or more of a list of complications were noted by the physician
Hypertension	Physician-identified hypertension
Preeclampsia	Physician-identified preeclampsia
CV–renal disease	Physician-identified cardiovascular–renal disease
Prepregnancy weight	Physician-reported maternal weight before pregnancy

[a] Variables are dichotomous (0 = no; 1 = yes), except as indicated.

[b] Compared with Hispanic White (the omitted category).

[c] The NNS and NFMS collapsed the top values to 17 or more years.

[d] Included as controls in the regressions of blood testing, advice about salt restriction, and advice about diuretics usage.

[e] The NNS and NFMS collapsed the top values to 30 or more visits.

[f] Included as controls in the regressions of advice about salt restriction and diuretics usage.

given by physicians regarding salt restriction and diuretics usage are compared with these expert recommendations.

Taffel and Keppel,[11] using the 1980 NNS, examined advice given by physicians about weight gain. They found that the majority of married mothers were given no weight gain limit or were advised to gain less than the amount currently recommended by nutritional experts. The advice varied according to race, educational background, parity, and prepregnancy weight.

Methods

Data

The National Center for Health Statistics based the NNS and NFMS on a complex random sample of 9941 live births and 6386 late fetal deaths that occurred in the United States during 1980.[12] Late fetal deaths are defined as fetal deaths with gestations of 28 weeks or more, or delivery weights of 1000 g or more. To adjust for the oversampling of fetal deaths, deflated sampling weights were attached to all observations for this research, resulting in a nationally representative sample. (Deflated sampling weights were calculated as the original sample weight times the sample size divided by the sum of weights.) The 2.3% of women who made no prenatal visits were excluded from these analyses.

Unimputed data were used from the live birth and fetal death records and from the follow-back questionnaires to the providers of prenatal care and the administrators of the hospitals where deliveries occurred. The prenatal care providers and the hospital administrators (hereafter referred to as the medical source) were asked to refer to medical records to answer detailed questionnaires about the prenatal care given to the women. The NNS and NFMS data have been described in detail elsewhere.[12,13]

Missing medical data may be due to the medical record being incomplete or invalid or it may be that the survey respondent did not fully transcribe the medical record. Donabedian argued that medical record keeping is one dimension of the quality of performance as well as the medium for evaluating other dimensions of care.[14] Also, two sources were asked to transcribe the medical information. Because of these factors, missing data were counted as missing examinations.

Variables

For the entire prenatal period, the medical sources recorded whether hematocrit and hemoglobin levels were obtained. For each prenatal visit, the medical sources recorded blood pressure and result of the urine test for protein. These data were transformed into the percentage of prenatal visits at which blood pressure was measured and the percentage of prenatal visits at which urine was checked for protein. The medical sources were also asked whether physicians advised women to restrict salt or to use diuretics during pregnancy.

The percentage of visits at which urine was tested is skewed to both ends of the range. Therefore, the variable was transformed into a dichotomous variable: whether urine was tested at no visits or at some percentage of visits greater than zero.

Table 1 lists the explanatory variables used in the analyses. The variables, "graduate" and "top visits" are indicator variables representing the top values of the "education" and "number visits" variables. "Graduate" and "top visits" were tested for an effect in each model but were only left in if they improved the model fit (Table 1).

The month of pregnancy that prenatal care was begun and the number of visits that the woman made were entered as controls in the blood test and advice models (Table 1). A provider does not expect to check hemoglobin and hematocrit levels or give particular pieces of advice more than a couple of times and may wait until

TABLE 2—Distribution of Prenatal Care Quality Variables

	Percentage Women
% of visits at which blood pressure was recorded	
None	12.9
1–24	3.5
25–99	16.4
100	67.2
% of visits at which urine tests were recorded	
None	33.7
1–49	6.3
50–99	35.2
100	24.8
Hemoglobin or hematocrit	
Not tested	6.6
Tested	93.4
Salt restriction	
Advised	14.9
Not advised	85.1
Diuretics usage	
Advised	2.6
Not advised	97.4

TABLE 3—Multiple Linear Regression of the Percentage of Visits at Which Blood Pressures Were Recorded on Maternal Sociodemographic Factors

Covariate	Regression Coefficient	Standard Error	Significance Level
Age	0.096	0.070	
Education	0.687	0.146	***
Non-Hispanic White	9.781	2.124	***
Black	−0.168	2.236	
Native American	1.615	3.683	
Asian/Pacific Islander	9.544	3.171	**
Married	4.040	0.918	***
Parity	−1.106	0.235	***
Metropolitan	−1.748	0.623	**

Note: F value = 44.029***; weighted frequency = 13 915. Covariates explained in Table 1.
**$P < .01$.
***$P < .001$.

TABLE 4—Multiple Logistic Regression of Urine Testing for Protein and Sugar on Maternal Sociodemographic Factors

Covariate	Regression Coefficient	Standard Error	Significance Level
Age	0.007	0.004	
Education	0.049	0.010	***
Graduate	0.061	0.108	
Non-Hispanic White	0.644	0.121	***
Black	0.070	0.127	
Native American	0.044	0.209	
Asian/Pacific Islander	0.456	0.187	*
Married	0.225	0.054	***
Parity	−0.071	0.014	***
Metropolitan	−0.085	0.038	*

Note: Likelihood ratio model χ^2 = 425.36 ***; weighted frequency = 14 155; urine never tested (0) = 4480, urine tested (1) = 9675. Covariates explained in Table 1.
*$P < .05$.
***$P < .001$.

after the first visit to do so. If the woman does not return for further visits after the first, or skips the visit when the test or counseling was scheduled, she may be less likely to receive these services at all.

The maternal health status factors listed in Table 1 were included as controls in the advice models because of their possible relationship with the sociodemographic and advice variables.

Statistics

Multiple linear regression analysis as implemented on the Statistical Analysis System (SAS)[15] was done in order to determine the statistical significance of sociodemographic factors in explaining the level of blood pressure monitoring during prenatal care. Logistic regression analysis using SAS[16] was used to estimate the effect of the sociodemographic factors on the occurrence of urine testing, blood testing, and the giving of appropriate advice.

Results

Prenatal Surveillance

During 1980, nearly one third of the women who obtained prenatal care (32.8%) did not receive blood pressure examinations at all of their prenatal visits. Providers skipped all blood pressure examinations on 12.9% of prenatal care recipients (Table 2). Over three quarters of women did not have urine tests for protein at all of the visits they made. One third of prenatal care recipients had no urine tests done during prenatal visits (Table 2). Prenatal care providers obtained no blood test for hemoglobin or hematocrit on 6.6% of the women who were prenatal care recipients (Table 2).

The set of sociodemographic factors (i.e., age, education, race or ethnicity, marital status, parity, and county of residence) explains only a minimal amount of the variance in the percentage of visits at which blood pressures were taken (R^2 = .027).[17] Nonetheless, most of the sociodemographic factors are statistically significant in the regression. For each additional year of education, there is an increase of two thirds of a percent in the number of visits at which blood pressure was taken. Non-Hispanic Whites and Asians and Pacific Islanders received blood pressure exams at almost 10% more of their visits than Hispanic White women (the excluded group). Native American and Black women did not differ in a statistically significant way from Hispanic White women. Married women had blood pressures taken at 4% more of their prenatal visits than unmarried women. For each additional level of parity, women had blood pressure checked at 1% fewer of the visits they made. Finally, women living in metropolitan counties had blood pressures taken at almost 2% fewer of the visits than did women living in nonmetropolitan counties (Table 3).

Turning to the urine test regression, the model χ^2 statistic shows that the set of sociodemographic variables are useful in classifying women in the test or no-test groups ($P < .001$).[17] The maximum likelihood estimate χ^2 statistic for the individual parameters tests the hypothesis that the parameter equals zero. According to this test, women with higher education

TABLE 5—Multiple Logistic Regression of Blood Testing for Hemoglobin (Hgb) or Hematocrit (Hct) on Maternal Sociodemographic Factors

Covariate	Regression Coefficient	Standard Error	Significance Level
Age	0.018	0.008	*
Education	0.013	0.183	
Graduate	1.349	0.393	***
Non-Hispanic White	0.211	0.224	
Black	−0.001	0.233	
Native American	−0.295	0.343	
Asian/Pacific Islander	0.563	0.433	
Married	0.552	0.094	***
Parity	−0.114	0.025	***
Metropolitan	−0.123	0.074	

Note: Likelihood ratio model χ^2 = 286.59***; weighted frequency = 14 155; Hgb or Hct not tested (0) = 869, Hgb or Hct tested (1) = 14 155. Control variables are month prenatal care began, number visits, and top visits. Covariates are explained in Table 1.

*$P < .05$.

***$P < .001$.

TABLE 6—Multiple Logistic Regression of the Advice Regarding Salt Restriction Given by the Physician on Maternal Factors

Covariate	Regression Coefficient	Standard Error	Significance Level
Age	−0.000	0.397	
Education	0.015	0.017	
Graduate	−0.155	0.161	
Non-Hispanic White	0.253	0.280	
Black	0.160	0.294	
Native American	−0.209	0.470	
Asian/Pacific Islander	0.007	0.386	
Married	0.297	0.109	**
Parity	−0.058	0.026	*
Metropolitan	−0.222	0.062	***

Note: Likelihood ratio model χ^2 = 364.99***; weighted frequency = 7577; salt restricted (1) = 1407, not restricted (0) = 6169. Control variables are month prenatal care began, number visits, top visits, hypertension, preeclampsia, % high BPs, and pre-pregnant wt. Covariates are explained in Table 1.

*$P < .05$.

**$P < .01$.

***$P < .001$.

were more likely than women with less education to receive urine tests at their prenatal visits. This was also true for non-Hispanic Whites and Asians or Pacific Islanders relative to Hispanic Whites. Married women were more likely to be tested at visits than unmarried women. Lower parity women were more likely than high-parity women to receive urine testing. And again, nonmetropolitan dwellers were at an advantage over metropolitan dwellers (Table 4).

As shown in Table 5, the sociodemographic variables are useful in classifying women as recipients of the blood test for hemoglobin or hematocrit ($P < .001$). Of the independent factors, maternal age, a graduate education, marital status, and parity are statistically significant. The likelihood that a woman would receive a blood test increased with age, as it did for married women and women of lower parity. Women with 17 or more years of education received blood tests for hemoglobin or hematocrit significantly more often than less educated women (Table 5).

Advice Concerning Salt Restriction and Diuretics Usage

Physicians reported advising salt restriction for 34.5% of hypertensive women, 36.7% of preeclamptic women, and 13.4% of women who were free of any physician-identified complications. Fifteen percent of the overall sample were advised to restrict salt (Table 2).

As shown in Table 6, the set of sociodemographic, health status, and prenatal care factors is useful in classifying women according to the kind of advice that they received regarding salt restriction ($P < .001$). The sociodemographic attributes that decrease the log-likelihood of being advised to restrict salt are being unmarried, having higher parity, and residing in a metropolitan county.

The final logistic regression shows that the set of sociodemographic and control variables is useful in classifying women according to whether they received advice to use diuretics ($P < .001$). Physicians were less likely to advise the use of diuretics for women who had higher parity, were younger, more educated, unmarried, or living in metropolitan counties (Table 7).

Discussion

Although these data are somewhat dated, it was deemed essential to use a nationally representative sample to analyze this health policy topic. Prenatal care data from the National Maternal and Infant Health Survey, the more recent version of the NNS and NFMS, will not be available for analysis until early 1992.

These results point to the importance of including quality of care measures in examining the adequacy of prenatal care. Prenatal care is far from being of even minimally acceptable quality for many women who have made the effort to engage the prenatal care system. High proportions of women were not tested, or not regularly tested, for the basics in prenatal care: blood pressure, urine, hemoglobin, and hematocrit. The advice that many women received concerning salt restriction and the use of diuretics was not congruent with current practice.

Distribution of the tests done to monitor the health of pregnant women varied substantially according to sociodemographic factors, as did the kinds of prenatal advice given. However, the groups of women who were likely to receive poor surveillance were mainly different from the groups likely to receive poor advice. These inconsistencies mean that physicians who provided prenatal care that was of good quality in terms of basic surveillance frequently gave advice that did not follow current recommendations.

The explanation for these inconsistencies may lie in the large percentage of variation in quality that is left unexplained by these models (Table 3). The setting of the health care practice is one variable that could be important. For example, physicians practicing in academic medical centers may have been aware of and followed new advice guidelines before physicians

TABLE 7—Multiple Logistic Regression of the Advice Given by the Physician Regarding Diuretic Use on Maternal Predictors

Covariate	Regression Coefficient	Standard Error	Significance Level
Age	0.034	0.017	*
Education	−0.147	0.039	***
Graduate	−1.320	0.794	
Non-Hispanic White	0.457	0.779	
Black	0.383	0.813	
Native American	−6.385	21.748	
Asian/Pacific Islander	0.361	1.069	
Married	0.916	0.292	**
Parity	−0.220	0.067	**
Metropolitan	−0.336	0.156	*

Note: Likelihood ratio model χ^2 = 318.86***; weighted frequency = 7384; advised to use drug (1) = 203, not advised to use (0) = 7181. Control variables are month prenatal care began, top visits, any complications, hypertension, cardiovascular–renal disease, preeclampsia, % high BPs, and prepregnancy wt. Covariates are explained in Table 1.

*$P < .05$.

**$P < .01$.

***$P < .001$.

in private practice. At the same time, those busy centers may have been settings where surveillance was missed. Without downplaying these inconsistencies, it seems best to concentrate most upon the surveillance results. The theories about optimal behavior in the areas of salt and diuretics usage had undergone recent revision in 1980. Well-qualified physicians may have known, but been unconvinced, that the recent changes in recommendations about salt and diuretics usage were sound. As pointed out by Hemminki,[1] disregarding changes in medical guidelines may in fact serve the best interests of the patient. She gives the example of x-rays, which were recommended during pregnancy and then found to be dangerous and rescinded.

The variability in quality of care according to maternal sociodemographic attributes may be explained in two ways. First, physicians allow their clinical judgment to be significantly affected by the nonmedical characteristics of the woman they are treating. Second, women of different sociodemographic backgrounds are obtaining health services from providers of substantially different quality. Although these data contain no information on the provider of prenatal care, we know that unmarried women would have higher likelihood of being on Medicaid.

Overall, the groups found to be in a relatively deprived position in terms of surveillance are already known to be the groups who obtain care later in pregnancy and in apparently inadequate quantity. It is possible that socially disadvantaged women discover early in life the low quality of the health services provided for them and decide that the costs of making visits (e.g., child care, transportation, opportunity costs of time spent) exceed the benefits.

From a policy perspective, this finding means that the prenatal care problem in this country rests as much with the quality of care delivered to disadvantaged women in particular as with this group's failure to seek the care that is available. Several approaches to the problem are apparent. We need to ascertain, first, that protocols for prenatal care at public facilities match the ACOG guidelines and, second, that these protocols are being followed by physicians and hospital staff. Third, we need to develop a quality control system for private providers. Federal and state funders of prenatal care should make quality assurance an integral part of their evaluation protocol. Finally, low reimbursement rates for prenatal care may contribute to the differences in surveillance outlined here. Providers of services to Medicaid recipients have an incentive to take shortcuts because of the low payment received for such services. Also, the training of the private providers of prenatal services to the poor may be poorer than that of providers to women with higher reimbursement potential.

On the other hand, women need information about the standard components of prenatal care. Most public education has been directed at the necessity for early prenatal care. The assumption has been that once the woman is integrated into the system, it will provide for her needs adequately. It is clear that for many women, this is an inaccurate portrayal of the situation. Basic information about the normal components of prenatal care could be incorporated in the educational campaign that promotes participation in the prenatal care system. Undoubtedly, well-informed and socially advantaged women not only choose better providers, but place demands on providers to deliver more services. Although women, especially socially disadvantaged women, cannot be expected to act as powerful enforcers of medical standards, they may at least be able to change providers if they are dissatisfied. □

Acknowledgments

I would like to thank the following people who provided helpful advice during the course of this research: Drs. Judith Blake, Virginia Flack, Carol Aneshensel, Calvin Hobel, Martin Shapiro, Paul Placek, and Carl Serrato. Honghu Liu assisted with the data analyses.

This study was supported in part by a Dissertation Research Grant from the National Center for Health Services Research and Health Care Technology Assessment (No. HS 06105).

An earlier draft of this paper was presented at the American Public Health Association 117th Annual Meeting in Chicago (October 25, 1989).

References

1. Hemminki E. Content of prenatal care in the United States: a historic perspective. *Med Care.* 1988;26:199–210.
2. Gold RB, Kenney AM, Singh S. *Blessed Events and the Bottom Line: Financing Maternity Care in the United States.* New York: The Alan Guttmacher Institute; 1987.
3. Institute of Medicine. *The Prevention of Low Birthweight: The Report of the Committee to Study the Prevention of Low Birthweight.* Washington, DC; 1985.
4. Kessner DM, Singer J, Kalk CE, Schlesinger ER. *Contrasts in Health Status. Infant Death: An Analysis by Maternal Risk and Health Care. I.* Washington, DC: Institute of Medicine; 1973.
5. Singh S, Torres A, Forrest JD. The need for prenatal care in the United States: evidence from the 1980 National Natality Survey. *Fam Plann Perspect.* 1985;17:118–124.
6. US General Accounting Office. *Prenatal Care. Medicaid Recipients and Uninsured Women Obtain Insufficient Care.* Washington, DC; 1987.
7. American College of Obstetricians and Gynecologists. *Standards for Obstetric-Gynecologic Services.* 5th ed. Washington, DC; 1982.
8. American College of Obstetricians and Gynecologists. *Standards for Ambulatory Obstetric Care.* Chicago, Ill; 1977.
9. Worley RJ. Pregnancy-induced hypertension. In: Danforth DN, Scott JR, eds. *Obstetrics and Gynecology.* 5th ed. Philadelphia, Pa: JP Lippincott Co; 1986:446–466.

10. Cruikshank DP. Medical and surgical complications of pregnancy: cardiovascular disease and neurologic disease. In: Danforth DN, Scott JR, eds. *Obstetrics and Gynecology*. 5th ed. Philadelphia, PA: JB Lippincott Co; 1986:492–502.
11. Taffel SM, Keppel KG. Advice about weight gain during pregnancy and actual weight gain. *Am J Public Health*. 1986; 76:1396–1399.
12. National Center for Health Statistics and Keppel KG, et al. *Methods and Response Characteristics, 1980 National Natality Survey, and Fetal Mortality Survey*. Washington, DC: Government Printing Office; Vital and Health Statistics Series 2(100)DHHS publication (PHS)86-1374.
13. National Center for Health Statistics. *Public Use Data Tape Documentation: National Natality Survey, 1980* and *National Fetal Mortality Survey, 1980*. Hyattsville, Md; 1983.
14. Donabedian A. Evaluating the quality of medical care. Milbank Q. 1966;44:166.
15. SAS Institute. *SAS User's Guide: Statistics, Version 5*. Cary, NC; 1985.
16. Harrell, FE. *SAS Supplemental Library User's Guide*. Cary, NC: SAS Institute; 1980:181–201.
17. Affi AA, Clark V. *Computer-Aided Multivariate Analysis*. Belmont, Calif: Lifetime Learning Publications; 1984.

Sixth National Conference on Chronic Disease Prevention and Control to Be Held in Washington, DC

The Centers for Disease Control, the Association of State and Territorial Health Officials, and the Association of State and Territorial Chronic Disease Program Directors are the cosponsors of the Sixth National Conference on Chronic Disease Prevention and Control entitled "Making Prevention a Reality." The conference will be held October 22 to 24, 1991, in Washington, DC, and is open to the public

The conference will emphasize interactions between federal, state, and local health departments; voluntary health agencies; and professional organizations. It will include two plenary sessions: Legislative Issues in Chronic Disease Prevention, and Community Action in Minority Communities for Achieving the Year 2000 Objectives. Concurrent sessions of invited papers will focus on the following topics:

- Use of Epidemiology for Program Decision Making
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- Breast and Cervical Cancer Control
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Effects of Paternal Occupational Exposure on Spontaneous Abortions

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ABSTRACT

Background: Paternal exposure to mutagenic agents has been suggested to affect pregnancy outcome adversely.

Methods: A nationwide data base of medically diagnosed spontaneous abortions and other pregnancies and national census data was used to evaluate the effects of men's occupational exposures on risk of spontaneous abortion in 99 186 pregnancies in Finland. Census data from the years 1975 and 1980 provided information about the occupation, industry, and socioeconomic status. A job-exposure classification was developed to classify women and their husbands according to possible occupational exposures on the basis of their occupational title and industry.

Results: In 10% of the pregnancies, the husband was exposed to one or more of the mutagens, and the rate of spontaneous abortion was unaffected (OR = 1.0). Of the 25 specific mutagenic exposures evaluated, paternal exposure to four (ethylene oxide, rubber chemicals, solvents used in refineries, and solvents used in the manufacturing of rubber products) was associated with an increased relative risk of spontaneous abortion. In addition, the risk of spontaneous abortion was higher among wives of rubber products workers than among unexposed men.

Conclusions: Although there is some biological rationale for the findings of this study, these findings need to be confirmed by studies in which individual exposures can be measured directly. (*Am J Public Health*. 1991;81:1029–1033)

Introduction

In animals paternal exposure to mutagenic compounds can increase the rate of spontaneous abortions.[1] In humans, however, this relationship remains unclear. For vinyl chloride,[2] anesthetic gases,[3] dibromochloropropane,[4] chloroprene,[5] smelter work,[6] waste water exposures,[7] and organic solvents,[8] effects have been suggested, but the data either have been contradictory or remain unconfirmed. An association has also been found with some paternal occupations: metal-plate and constructional steel workers, crushers and grinders, sewers, workers caring for fur-bearing animals,[9] and mechanics and repairers of motor vehicles.[10]

Several methodologic issues must be considered in conducting studies concerning male effects on reproduction. First, such studies must take into account maternal factors. Second, they should collect data on pregnancy outcomes which minimize systematic bias. Third, they need large enough numbers of exposed men to provide reasonable statistical power. We attempted to address these issues in studying the effects of male occupational exposures on the outcome of pregnancy by using data from two national censuses and a nationwide data base of medically diagnosed pregnancy outcomes. Exposures were determined indirectly by using a job-exposure classification. This large dataset can be used to generate hypotheses for other studies of the effects of occupational exposures on reproductive outcomes.

Methods

Data on reproductive outcomes and maternal age were obtained from the nationwide Hospital Discharge Register (HDR) and from hospital clinics. We collected data on all women with a diagnosis of spontaneous abortion (ICD-8 codes 643 and 645), induced abortion (ICD-8 codes 640–642) and birth (ICD-8 codes 650–662) between 1973 and 1982. A separate questionnaire was sent to the hospitals to collect information on patients treated on an outpatient basis and not registered in the HDR. The content, technical quality, and reliability of the data base of medically diagnosed pregnancies have been described elsewhere.[11]

Information on the occupation and industry of women and their husbands and the women's socioeconomic status was obtained from the 1975 and 1980 national censuses of the Central Statistical Office of Finland. Pregnancy data for the years 1973 to 1978 were linked to the 1975 census, and the data for the years 1979 to 1982 to the 1980 census.

In the census of 1975, performed on December 31, 1975, occupational information was requested as follows: "Occupation in the place of employment" with the instruction "State your occupation as accurately as possible, not only by hon-

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This paper was submitted to the journal March 6, 1990, and accepted with revisions February 15, 1991.

TABLE 1—Pregnancy Outcomes Among Wives and Adjusted[a] Odds Ratio of Spontaneous Abortion for Paternal Exposure to Mutagenic Agents

Husband's Exposure Status	Spontaneous Abortions	Induced Abortions	Births	All Pregnancies	Odds Ratio	95% Confidence Interval
Not exposed[b]	7772	7029	72 8[illegible]	87 616	1.0	
Potential, low exposure	820	1045	806[illegible]	9930	1.0	0.9 – 1.0
Moderate or high exposure	139	151	1350	1640	1.0	0.9 – 1.2

[a] Adjusted for age (≤24, 25–29, 30–34, 35–39, ≥40); socioeconomic status (1 = employers, own-account workers, upper level employees, 2 = lower level employees, students and pupils, 3 = manual workers); maternal exposure to reproductive hazards (not exposed, exposed).

[b] Referent category.

orary titles or degrees." In the census of 1980, performed on November 1, 1980, the question was, "In which occupation did you work for the longest period during the last 12 months?" Occupations were coded by census officials, on the basis of the Nordic Classification of Occupations and the International Standard Classification of Occupations. Industry was coded on the basis of the Finnish Standard Industrial Classification.

We classified the occupational exposure status of men and women on the basis of occupation and industry, using a job-exposure classification developed in cooperation with two experienced industrial hygienists. The job-exposure classification is organized by job groups, which are combinations of occupation and industry having a similar type of exposure. For each job group a list is given of agents suspected of having adverse effects on reproduction. The assessment of the exposures among the job groups was based on the industrial hygienic measurements made by the Institute of Occupational Health[12,13] and the Finnish register of employees occupationally exposed to carcinogens (ASA Register).[14]

The list of potentially dangerous agents in the classification comprises 78 exposures, including separate chemical substances (e.g., benzene, ethylene oxide), mixtures of exposures (e.g., mixtures of solvents or metals), and nonspecific exposure groups (e.g., biologic factors, rubber chemicals, pesticides). Twenty-five of the 78 exposures were suspected mutagens, based mainly on the classification of the International Agency for Research on Cancer.[15]

Three levels of exposure were distinguished according to the results of industrial hygienic measurements and the prevalence of exposure in the job group: "moderate or high," "potential, low," and "none." "Moderate or high exposure" included jobs in which the level of exposure to mutagens was continuously at least half of the Threshold Limit Value (TLV) or higher or in which the exposure periodically exceeded TLV and the prevalence of exposure was high. "Potential, low exposure" denoted either (a) jobs with low level but high prevalence of exposure to mutagens, (b) jobs which lacked industrial hygiene measurements but which were reported to the ASA Register, or (c) jobs with a high level and unknown prevalence of exposure.

The susceptible time period was determined on the basis of the time of conception and the time period for which the two censuses obtained information on occupation. For men, the time of spermatogenesis (80 days before conception) was defined as the relevant time period for exposure; for women, the relevant time period was considered to be the first trimester of pregnancy. The analysis of exposure to any mutagenic agents was limited to the pregnancies terminated between January 1, 1976, and December 31, 1976, and between May 1, 1980, and April 30, 1981. In the analysis of specific mutagenic exposures we used a 2-year period close to the census, i.e., January 1, 1976, to December 31, 1977, and May 1, 1980, to April 30, 1982, because the number of workers exposed to a specific agent was often small.

After the pregnancies of women less than 12 and more than 50 years of age had been excluded as well as those with unspecific data on men's and women's occupation, industry, or socioeconomic status, the study population included in the analysis for the limited time periods of January 1 to December 31, 1976, and May 1, 1980 to April 30, 1981, comprised 99 186 pregnancies.

The risk odds ratios comparing the odds of spontaneous abortion for paternal exposure to a mutagen with the odds of spontaneous abortion for the nonexposed group were obtained by using linear logistic regression analysis.[16] To minimize random error in the effect estimates, low and moderate or high levels of exposure were combined if the number of the workers in either category was too small. Age, socioeconomic status, and maternal exposure to potential reproductive hazards were included in the models to control for the confounding effect of these variables. To gain precision, odds ratios were only adjusted for age if fewer than 100 pregnancies occurred among the workers exposed to a chemical. The exclusion of socioeconomic status in the model did not significantly change the odds ratios for these exposures. Maximum likelihood estimates of odds ratios, which were used as surrogate measures of relative risks, and their 95% confidence intervals were calculated by using the GLIM computer program.[17] Odds ratios for exposure to mutagens were similar regardless of whether induced abortions were included or excluded from the denominator. In calculating the rate of spontaneous abortion, we included births, spontaneous abortions, and induced abortions in the denominator. (Although it is common in the literature to refer to these measures as rates, they are in fact proportions or risks because they are calculated by dividing the number of spontaneous abortions by the total number of pregnancies.[16])

Results

The rate of spontaneous abortion in the study population was 8.8%, which is nearly the same as the rate among all Finnish women (8.9%).[11] Of the husbands, 10% were considered to have low or potential occupational exposure to mutagenic agents and only 1.7% were classified as heavily exposed (Table 1). After adjusting for maternal age, socioeconomic status, and maternal exposure to reproductive hazards, the odds ratio of spontaneous abortion did not differ significantly from one for either level of paternal exposure to mutagens (Table 1).

The adjusted odds ratio was significantly increased for four paternal exposures: ethylene oxide (OR = 4.7, 95% CI 1.2-18.4), solvents used in the petroleum refineries (OR = 2.2, 95% CI 1.3-3.8), rubber chemicals (OR = 1.5, 95% CI 1.1-2.2) and a subgroup of the former, solvents used in the manufacture of rubber products (OR = 1.9, 95% CI 1.2-2.8; Table 2). Except for ethylene oxide, these exposures are mixtures of several chemicals. From the solvents used in the refineries, gasoline and benzene are suspected to be mutagens. Similarly, from the solvents used in the manufacture of rubber products 1,1,1-trichloroethane and methylene chloride are suspected mutagens. Some of these chemicals also appear in the classification as specific exposures. When paternal exposure to gasoline, benzene, and 1,1,1-trichloroethane were analyzed separately, the odds ratios did not differ significantly from unity.

The risks of spontaneous abortions were also calculated for paternal exposure to lead, carbon disulfide, and heat, which are not mutagens but may adversely affect spermatogenesis. For all these exposures, the odds ratio was below unity but the corresponding confidence interval did not exclude one (Table 3).

In some cases, job title may be a good and specific index of a worker's exposure. For this reason, we calculated the odds ratios of spontaneous abortion by paternal occupation using the 55 occupations included in the exposure classification. The comparison group consisted of wives whose husbands were not exposed to any mutagen. Table 4 lists in descending order the paternal occupations with the highest odds ratios (> 1.20).

Only the results obtained for wives of men employed as rubber product workers reached statistical significance. At the top of the list are also two other occupations in which workers may be exposed to chemicals which we found were associated with an increased risk of abortions (Table 2). These occupations are technical nursing assistants (potentially exposed to ethylene oxide) and oil refinery workers (potentially exposed to solvents of oil manufacture). Other occupations with a high risk of abortion were watchmakers and chimney sweeps.

Discussion

Paternal exposure to certain mutagens may induce spontaneous abortions. We found no difference in the risk of spontaneous abortion between wives of men exposed to any mutagen and wives of men exposed to no mutagen. This may suggest that only a subgroup of mutagens induce spontaneous abortions. Moreover, the mutagenicity of some of the classified agents, such as 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, chloroform, methylene chloride, and the

TABLE 2—Adjusted[a] Odds Ratio of Spontaneous Abortion for Paternal Exposure to Specific Mutagens[b]

Exposure[c] (Level of Exposure)	Number of Spontaneous Abortions	Number of Pregnancies	Odds Ratio	95% Confidence Interval
Ethylene oxide (low)	3	10	4.7*[d]	1.2–18.4
Solvents, petroleum refineries (gasoline, benzene) (moderate or high)	16	93	2.2**[d]	1.3–3.8
Impregnents of wood (arsenic, chromium, creosotes) (low and moderate or high)	5	33	1.9[d]	0.7–5.0
Solvents, manufacture of rubber products (1,1,1-trichloroethane, methylene chloride) (low and moderate or high)	26	180	1.9**	1.2–2.8
Rubber chemicals (low and moderate or high)	34	280	1.5*	1.1–2.2
1,1,1-Trichloroethane (moderate or high)	3	24	1.5[d]	0.4–5.0
Radon (moderate or high)	26	248	1.2	0.8–1.8
Formaldehyde (formaline)				
Low exposure	110	1212	1.1	0.9–1.4
Moderate or high exposure	54	596	1.0	0.8–1.4
Polycyclic aromatic hydrocarbons				
Low exposure	1022	11 970	1.0	0.9–1.0
Moderate or high exposure	142	1619	1.0	0.9–1.2
Nickel and nickel oxides (low)	284	3371	1.0	0.9–1.1
Chromium and chromium compounds (low)	286	3444	1.0	0.9–1.1
Solvents, manufacture of plastic products (styrene) (moderate or high)	24	285	1.0	0.7–1.5
Benzene (low)	55	655	1.0	0.7–1.3
Metals (nickel, chromium, cadmium), basic metal industries and manufacture of metal products (low)	155	1803	1.0	0.8–1.1
Motor gasoline (low)	189	2381	0.9	0.8–1.1
Styrene (moderate or high)	9	120	0.9	0.5–1.8
Trichloroethylene (low and moderate or high)	5	66	0.9[d]	0.3–2.1
Plastic monomers (low)	10	158	0.8	0.4–1.4
Tetrachloroethylene (moderate or high)	3	45	0.7[d]	0.2–2.4
Solvents, manufacture of drugs (chloroform, methylene chloride) (moderate or high)	3	60	0.5[d]	0.2–1.7
Raw materials of glass (arsenic, cadmium) (low)	4	99	0.5[d]	0.2–1.3
Hydrazine (low)	2	51	0.4[d]	0.1–1.7
Solvents, manufacture of printing inks (ethene) (moderate or high)	1	33	0.3[d]	0.0–2.5
Nitroglycol, nitroglyserine, trinitrotoluene (low)	1	34	0.3[d]	0.0–2.3
Solvents, manufacture of glue and gasein (ethene) (moderate or high)	1	35	0.3[d]	0.0–2.3

[a] Adjusted for age (≤24, 25–34, ≥35); socioeconomic status (1 = employers, own-account workers, upper level employees, 2 = lower level employees, students and pupils, 3 = manual workers).

[b] Comparison group was not exposed to any mutagen.

[c] In parentheses are the exposures suspected to be mutagens.

[d] Adjusted for age only.

*$P < .05$, **$P < .01$

TABLE 3—Adjusted[a] Odds Ratio of Spontaneous Abortions for Exposure to Lead, Carbon Disulfide, and Heat[b]

Exposure (Level of Exposure)	Number of Spontaneous Abortions	Number of Pregnancies	Odds Ratio	95% Confidence Interval
Lead (low and high)	352	4476	0.9	0.8–1.0
Carbon disulfide (high)	2	30	0.8[c]	0.2–3.3
Heat (low)	4	109	0.4[c]	0.2–1.2

[a] Adjusted for age (≤24, 25–34, ≥35); socioeconomic status (1 = employers, own-account workers, upper level employees, 2 = lower level employees, students and pupils, 3 = manual workers).
[b] Comparison group was not exposed to any mutagen, lead, carbon disulfide, or heat.
[c] Adjusted for age only.

TABLE 4—Adjusted[a] Odds Ratio of Spontaneous Abortions in the Occupations of the Exposure Classification with Odds Ratio > 1.20[b]

Occupation	Spontaneous Abortions	Pregnancies	Odds Ratio	95% Confidence Interval
Technical nursing assistants	3	22	1.6	0.5–5.6
Rubber product workers	35	281	1.5[*]	1.1–2.2
Watchmakers	13	102	1.5	0.8–2.7
Chimney sweeps	18	141	1.5	0.9–2.4
Refinery workers	13	111	1.4	0.8–2.5
Smelters, hardeners, temperers in metal industry	4	37	1.4	0.5–3.9
Textile finishers and dyers	14	130	1.3	0.8–2.4
Wire and pipe drawers	5	50	1.2	0.5–3.1
Textile machine setters, operators	22	217	1.2	0.8–1.9

[a] Adjusted for age (≤24, 25–34, ≥35 years).
[b] Comparison group was not exposed to any mutagen.
[*] $P < .05$.

metals as a group is not well established and depends on the mutation assay used. Thus mutagenicity of the agents such as these is debatable.[15,18] Another example of lack of effect is paternal smoking, one of the most common mutagenic exposures, which appears to have no effect on fertility (time to pregnancy).[19,20]

Of the 25 specific mutagens or groups of mutagens evaluated in this study, paternal exposure to the following four was associated with an increased risk of spontaneous abortion: ethylene oxide, rubber chemicals, solvents used in petroleum refineries, and solvents used in the manufacture of rubber products. Among these, ethylene oxide has been identified as a mutagen by almost all mutation assays, including the dominant lethal assay.[15] Exposure to this chemical has also been associated with spontaneous abortion in women who use the chemical to sterilize hospital instruments.[21] However, our finding of the effects of ethylene oxide is based on a small group of exposed workers. In addition, the job group to which exposure to ethylene oxide was attributed is heterogeneous, and no individual exposures could be confirmed. Rubber chemicals contain several microbial mutagens,[22] and an increased risk of abortion has been observed among women exposed to rubber chemicals.[23] An excessive rate of spontaneous abortion has been found among the wives of workers in a waste water treatment plant of a petroleum refinery[7] and among the wives of workers exposed to organic solvents.[8]

Analyses by occupational title identified only 1 out of 55 occupations, rubber workers, for which the wife's risk of spontaneous abortions was higher than that of wives of men not exposed to mutagens. The wives of men exposed to potentially spermatotoxic but nonmutagenic agents, such as lead, carbon disulfide and heat, did not appear to have an increased risk of spontaneous abortions.

Genetic damage to male germ cells is the only mechanism of male-mediated effects on pregnancy outcome for which supporting evidence is available.[24] Another potential mechanism could be that harmful substances are transmitted to the pregnant woman by contact with clothes or by semen leading to secondary maternal exposure. We were unable to separate the effects of these two routes of exposure.

The misclassification of exposure and pregnancy outcome is an important source of bias in studies of occupational exposure and reproductive outcomes. In this study, misclassification of pregnancy outcome was likely to be small because the data were obtained from hospital records, and spontaneous abortions were medically diagnosed. These data were therefore less susceptible to the information bias which might arise from self-reported data on pregnancy outcome.[11]

In Finland induced abortions are legal. If any induced abortions are reported as spontaneous abortions, we would expect this misclassification to be very small.

It is certain that early spontaneous abortions and a small percentage of induced abortions were not included. The duration of pregnancy before a spontaneous abortion may vary by paternal exposure status. If early fetal losses, which were not included in the study, occurred more often among wives of men exposed to mutagens, the odds ratios reported would underestimate the true relative risk. If the converse were true, the odds ratios would overestimate the true relative risk.

The weaknesses of the study pertain to the potential misclassification of exposure status. Assignment of exposure status was made on the basis of occupational titles and industry. In some cases, these data may not have been sufficiently specific to assess exposure status accurately. Consequently, some exposed persons may have been classified incorrectly as unexposed, and this could have resulted in low sensitivity of the exposure assessment. Conversely, occupational categories may have been so broad that unexposed persons may have been classified as exposed, inducing low specificity. Random error in the classification of exposure status (nondifferential misclassification) may have diluted the effects of exposures and may explain the absence of associations in this study.

Although census data do not provide completely accurate exposure information, care was taken to assess exposure as accurately as possible. First, analyses were carried out in the time period close to the date of the censuses to allow minimal time for change in occupation. Second, exposure status was determined independently of reproductive outcome. Third, the job exposure classification rather than mere job title was used to assign exposure status; in some cases it was possible to quantify the exposure.

No information was available on other potential confounding factors such as previous spontaneous abortions or the use of alcohol or smoking. Probably the adjustment of the socioeconomic status has partly controlled the effect of smoking and alcohol drinking, as they vary by social class.

This large study showed no overall excess of spontaneous abortions in women whose husbands were exposed to mutagens in general compared with those whose husbands were not exposed to mutagens. However, excesses were observed among wives of men employed as rubber products workers and of men exposed to ethylene oxide, solvents used in petroleum refineries, rubber chemicals, and solvents used in the manufacturing of rubber products. Many comparisons were made in this study, and some of the associations probably reflect chance alone. Thus, our findings are only suggestive. This emphasizes the need to base inferences on biologic plausibility and on the evidence accumulated from this and other studies. Although there is some biological rationale for these findings, they need to be confirmed by studies in which individual exposures can be assessed directly. ☐

Acknowledgments

This project was funded in part by Family Health International under a cooperative agreement with the United States Agency for International Development. However, the views expressed in this article do not necessarily reflect those of the funding agency. Family Health International is an international not-for-profit organization which conducts research and provides technical assistance in health, family planning, sexually transmitted diseases, and AIDS.

The authors wish to thank Mr. Robert Lloyd and Mr. Pekka Pulkkinen for computer programming work and Ms. Tarja Nikula for technical assistance in data management.

References

1. Epstein SS, Arnold E, Andrea J, Bass W, Bishop Y. Detection of chemical mutagens by the dominant lethality assay in the mouse. *Toxicol Appl Pharmacol*. 1972;23:288–325.
2. Infante PF, Wagoner JK, McMichael AJ, Waxweiler RJ, Falk H. Genetic risks of vinyl chloride. *Lancet*. 1976;1:734.
3. Tomlin PJ. Health problems of anaesthetists and their families in the West Midlands. *Br Med J*. 1979;1:779–784.
4. Kharrazi M, Potashnik G, Goldsmith JR. Reproductive effects of dibromochloropropane. *Isr J Med Sci*. 1980;16:403–406.
5. Sanotsky IV. Aspects of the toxicology of chloroprene: Immediate and longterm effects. *Environ Health Perspect*. 1976;17:85–93.
6. Beckman L, Nordström S. Occupational and environmental risks in and around a smelter in Northern Sweden. IX. Fetal mortality among wives of smelter workers. *Hereditas*. 1982;97:1–7.
7. Morgan RW, Kheifets L, Obrinsky DL, Whorton MD, Foliart DE: Fetal loss and work in a waste water treatment plant. *Am J Public Health*. 1984;74:499–501.
8. Taskinen H, Anttila A, Lindbohm M-L, Sallmén M, Hemminki K. Spontaneous abortions and congenital malformations among the wives of men occupationally exposed to organic solvents. *Scand J Work Environ Health*. 1989;15:345–352.
9. Lindbohm M-L, Hemminki K, Kyyrönen P: Parental occupational exposure and spontaneous abortions in Finland. *Am J Epidemiol*. 1984;120:370–378.
10. McDonald AD, McDonald JC, Armstrong B, Cherry NM, Nolin AD, Robert D. Fathers' occupation and pregnancy outcome. *Br J Ind Med*. [London]. 1989;46:329–333.
11. Lindbohm ML, Hemminki K. Nationwide data base on medically diagnosed spontaneous abortions in Finland. *Int J Epidemiol*. 1988;17:568–573.
12. Skyttä E. *Tilasto työhygieenisistä mittauksista v. 1971–1976*. Helsinki: Työterveyslaitos Katsauksia 17;1978. (In Finnish)
13. Kokko A. *Tilasto työhygieenisistä mittauksista v. 1977–1980*. Helsinki: Työterveyslaitos Katsauksia 59;1982. (In Finnish)
14. Heikkilä P, Savela A, Vuorela R, Partanen T. *Employees exposed to carcinogens in Finland in 1986.* Reviews 13. Helsinki: Institute of Occupational Health, 1988.
15. International Agency for Research on Cancer. *Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs, Vols 1 to 42: Suppl 7.* Lyon, France: IARC; 1988. IARC Monographs on the evaluation of carcinogenic risks to humans.
16. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research. Principles and Quantitative Methods.* New York, NY: Van Nostrand Reinhold Company Inc; 1982.
17. Baker RJ, Nelder JA. *The GLIM System, Release 3.* Oxford, England: Royal Statistical Society; 1978.
18. International Agency for Research on Cancer. *Long-Term and Short-Term Screening Assays for Carcinogens: A Critical Appraisal.* Lyon, France: IARC; 1980. IARC Monographs, Suppl 2.
19. Rosenberg MJ. *Smoking and male fertility. Seminars in Reproductive Endocrinology.* November 1989;7(4):314–318.
20. Baird DD, Wilcox AJ. *Cigarette smoking associated with delayed conception.* JAMA 1985;253:2979–2983.
21. Hemminki K, Mutanen P, Saloniemi I, Niemi ML, Vainio H. *Spontaneous abortions in hospital staff engaged in sterilizing instruments with chemical agents. Br Med J.* 1982;285:1461–1463.
22. Donner M, Husgafvel-Pursiainen K, Jenssen D, Rannug A. Mutagenicity of rubber additives and curing fumes. *Scand J Work Environ Health*. 1983;9(Suppl 2):27–37.
23. Lindbohm ML, Hemminki K, Kyyrönen P, Kilpikari I, Vainio H. Spontaneous abortions among rubber workers and congenital malformations in their offspring. *Scand J Work Environ Health*. 1983;9(Suppl 2):85–90.
24. Fabro S: Paternally-induced adverse pregnancy effects. *Reprod Tox* 1984;3:13–16.

The Health Hazards of Saunas and Spas and How to Minimize Them

Edward Press, MD, MPH

ABSTRACT

Background: The rapidly increasing number of spas, hot tubs, and saunas intensifies the potentials for deaths from hyperthermia and drowning.

Methods: I analyzed 54 such deaths reported to me by 55 medical examiners and coroners in the United States and 104 deaths reported to the US Consumer Product Safety Commission (CPSC).

Results: Only seven of the 158 deaths analyzed occurred in saunas. All of the remaining deaths occurred in spas, jacuzzis, or hot tubs, which were far more numerous. The chief risk factors identified were alcohol ingestion, heart disease, seizure disorders, and cocaine ingestion (alone or in combination with alcohol ingestion). These factors accounted for 71 or 44.7% of the 159 fatalities. Of these risk factors, alcohol represented 38%; heart disease, 31%; seizure disorders, 17%; and cocaine ingestion, alone or in combination with alcohol, 14%. Sixty-one of the 151 spa-associated deaths occurred in children under 12 years of age. Accidental drownings from uncovered or improperly covered spas and,to a lesser extent, entrapment by suction, were the chief causes of childhood drownings.

Conclusions: Children and older persons who have heart disease or seizure disorders or who use alcohol or cocaine are especially vulnerable. Recommended preventive measures include shortening the time of exposure, lowering the temperature, establishing safety standards for covers and for baffles for suction outlets, and using warning notices. (*Am J Public Health*. 1991;81:1034–1037)

Introduction

The "dry" or Finnish Sauna is an enclosure large enough for two to eight or more persons; it generally has no active ventilation, is heated by electricity, gas, or other means; temperatures are maintained at 70°C to 90°C, but often can be raised up to 100°C. Spas are large bath tubs or small pools made of synthetic materials; they have equipment that heats, recirculates, and disinfects the water and usually has jets of water or air or both. Hot tubs are similar to spas, but they are made of wood and their recirculating water may or may not have jets of water or air. The water in both spas and hot tubs is heated to about 40°C, but may go even higher. Health hazards from hot tubs are similar to those from spas. In this article the term "spas" includes hot tubs, but does not include the smaller jetted bath tubs that are emptied after each use.

The exact number of saunas in the United States is not known. The Sauna Society of America estimates the number at about 1 million (personal communication from V.S. Choslowsky, President, Sauna Society of America, Sept. 1988). More accurate estimates are available for spas and hot tubs; one of the trade associations placed the number at about 1.8 million in 1988 (personal communication from Larry E. Paulick, Sr., vice-president, National Spa and Pool Institute, Alexandria, Va, Dec. 5, 1988). A trade publication placed the number that are sold annually at 250 000 (personal communication from Kathleen Bedenbaugh, editor, Aqua Magazine, Harcourt-Brace Jovanovich Publications, Santa Anna, Ca, Oct. 1988. Thus there would be millions of monthly exposures to the hazards of hyperthermia and drowning. To reduce the risks from these exposures, voluntary standards have been developed by several organizations. One of these, Underwriters Laboratories' Standard 875 for Electric Saunas requires the use of the following warning sign: "Do not exceed 30 minutes in sauna. Persons with poor health should consult physician before using sauna." Because (as noted below) a 30-minute exposure has been found to be unsafe for some persons, and because there is so little information available on mortality in saunas and spas in the United States, I undertook a literature review and then attempted to collect information on deaths associated with spas and saunas.

Literature Review

According to Kauppinen and Vuori,[1] warmth induces a feeling of euphoria, relaxation, and tranquility in many persons. Sorri discussed saunas in a regular issue of the Annals of Clinical Research[2] and in a special issue of the same journal,[3] which includes two editorials and 16 articles on the Finnish sauna. The author states that bathing in a sauna is a pleasant and relaxing experience, which combines psychic, physical, and social pleasures; reduces aggressive behavior; and enables bathers to forget the common pressures of everyday life. He also feels that the nature of the experience makes it difficult to define objective scientific methods of measurement.

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This paper was submitted to the journal February 27, 1990, and accepted with revisions February 19, 1991.

The increased pressure from the water results in peripheral vasodilation, profuse perspiration with a decrease in the extracellular blood volume, an increased viscosity of the blood, an increase in hematocrit with shortened clotting time, and a 20% increase in the consumption of oxygen.[1] The electrocardiographic (ECG) changes include a T-wave depression, ventricular and supraventricular extrasystoles, and a deep S wave.[4] Thus the possibility of cardiac arrest exists. This would be especially true for persons with preexisting heart disease or those on medications that influence cardiac rhythms. The combination of peripheral vasodilation and decreased blood volume from the loss of fluids reduces the cerebral blood flow and increases the likelihood of syncope. Three out of 60 subjects using a Finnish Sauna for 20 minutes at a temperature of 80°C to 90°C fainted; a fourth, a 63-year-old, apparently healthy, man, developed an angina attack; his ECG tracings showed ventricular and extraventricular extrasystoles, which persisted for a half hour after he left the sauna.[4] Jokinen et al.[5] studying children found that of 61 children that had a 10-minute exposure in a sauna, two developed vasovagal collapses.

Diabetics receiving subcutaneous injections of insulin may absorb their insulin much more rapidly because of the peripheral vasodilation. Khogali and Gales[6] feel that "it is quite common for patients with heat stroke to have diabetes, as though they are predisposed. . . . Since diabetics often exhibit postural hypotension due to faulty baroreflexes it is conceivable that this could be responsible for the relatively poor adjustments in vasculature of various body regions." Sweating abnormalities and neuropathy of the autonomic nervous system also are common to diabetics. Hormonal activity resulting in increased secretions of catecholamines and an increased sympathomimetic activity have been reported.

Because in the sauna and spa the testicles, which are normally below internal body temperature, are exposed to temperatures well above normal body temperature, the possibility of impaired spermatogenesis exists. B. J. Procope, as quoted by Kauppinen and Vuori[1] stated that intensive sauna bathing, repeated six to eight times within a 2-week period, caused a reduction in the number of sperm cells in 12 medical students accustomed to the sauna. Brown-Woodman, also quoted by Kauppinen and Vuori, reported that even a single 20-minute exposure to a sauna at 80°C decreased the number of sperm cells starting 1 week after the bathing and lasting for 5 weeks. He found that there were no changes in the morphology; but ultrastructural changes of the sperm cell were observed.

Questions of potential teratologic effects on the developing fetus when pregnant women are exposed to the hyperthermia of the sauna and the possibility that such exposure might induce labor prematurely in women approaching full term, have also been raised. Edwards[7] in a fairly extensive review of the clinical significance of experimental studies on hyperthermia as a teratogen, found that "the species known to be affected include birds, all the common laboratory animals, farm animals, and primates. It would be remarkable if it were not also a human teratogen." He concluded that because there is very strong evidence that hyperthermia is a human teratogen more study is required to fully determine the nature and extent of this risk to prenatal development. He thought that an elevation of 1.5°C to 2.5°C above the species' normal temperature could result in a heat-induced teratogenic effect.

Vaha-Eskeli and Erkkola[8] after reviewing some of the literature and testing 23 pregnant women, concluded that at least, as practiced in Finland, hyperthermia was not teratogenic. The authors did, however, comment that in Finland it is commonly believed that sauna bathing could be used to induce labor in postterm women. In their study of the effect of thermal stress on 23 women who were 36 weeks pregnant, they found that 5 of the women had occasional uterine contractions, although 1 of the 5 experienced the contractions before, as well as after, the sauna exposure.

Method

After a preliminary telephone poll of 16 US medical examiners and coroners, a questionnaire (available from the author) requesting information on the number and type of deaths associated with saunas, spas, and hot tubs and related risk factors noted during the past 5 years was sent to 184 members of the National Association of Medical Examiners. The members were selected on a subjective basis from the entire list of 654 members. When several different members were from the same office, the chief or director, rather than deputies, assistants, or consulting pathologists was chosen. In addition, wide geographic distribution coupled with representation from urban and rural areas and from hot and cold climates were factors in the selection. To supplement these factors, a medical examiner who was a longtime member of the association and familiar with many of its members personally chose about 10 or 12 whom he considered experienced and well trained.

A total of 55 responses were received. These were followed up by telephone interviews with 20 of the 55 for more complete and detailed information. Thirty-two of the 55 respondents indicated that they could remember no fatalities during the preceding 5 years (from November 1988).

The Consumer Product Safety Commission (CPSC) has a program called the Medical Examiners and Coroners Alert Program asking medical examiners and coroners to report deaths to the CPSC. Reports of such deaths in saunas and spas were obtained from the CPSC for the period of January 1, 1986 to November 4, 1988. The reports did not include all states in the United States for the entire period, nor did those reporting necessarily include all cases in their jurisdiction.

Results

Age distribution. Only 15% of the deaths reported in my survey (all in spas), were in children under 12 years of age, compared with 52% reported to the CPSC (Table 1). This discrepancy could reflect the fact that the reports to the CPSC were more recent, most of them prepared in 1987 and 1988 when a larger number of spas were sold to private residences than to public facilities and athletic clubs, which are much less likely to have children present. The author's poll went back 5 years from 1988 when the number of private spas was lower.

Duration of exposure. Ten of the 24 jurisdictions reporting fatalities in my survey recorded the length of exposure to the spa prior to death. In four cases the exposure was more than 30 minutes; in four more, it was 10 to 20 minutes; and in two it was less than 10 minutes.

Risk Factors. Risk factors were reported in 45% of the deaths (Table 2). Alcohol ingestion and heart disease were the chief factors associated with the fatalities. Seizure disorders and cocaine ingestion either alone, or cocaine combined with alcohol, accounted for additional risk factors. In many deaths the presence or absence of risk factors was either unknown or unrecorded.

TABLE 1—Age Distribution of Spa and Sauna Fatalities

	SPAS CPSC[a]		SPAS EP Survey[b]		SAUNAS EP Survey[b]	
Age	N	%	N	%	N	%
0–2 y	36	35%	2	4%	0	0%
2–11 y	18	17%	5	11%	0	0%
12–65 y	31	30%	29	62%	2	29%
over 65 y	19	18%	9	19%	5	71%
Unknown	0	0%	2	4%	0	0%
Total	104	100%	47	100%	7	100%

[a] US Consumer Product Safety Commission reports, Jan 1, 1985, to Nov 4, 1988.
[b] Reports to the author from 55 county and state medical examiners and coroners, Nov 1988.

TABLE 2—Risk Factors Reported as Associated with Spa and Sauna Deaths

	SPAS CPSC[a]	SPAS[b]	SAUNAS[b]	TOTAL
Alcohol	10	15	2	27
Heart Disease	10	8	4	22
Seizures	6	6		12
Cocaine	1	2		3
Cocaine & alcohol	3	4		7
Total Reported	30	35	6	71 (44.65%)[c]

[a] Consumer Product Safety Commission's death certificate reports from their Medical Examiners and Coroners Alert Program. In only 50 of the 104 deaths were the risk factors checked as present or unknown. In the others, no mention was made of them.

[b] Author's poll of 55 medical examiners or coroners. In this poll, obesity was recorded in association with heart disease or alcohol in 8 cases.

[c] $\frac{71}{104+55} \times 100 = 44.65\%$

Discussion

The chief physiological differences between spas and saunas relate to the more rapid transfer of heat from water than from air. The jet currents in the spa further enhance the heat transfer and the evaporative cooling effects of perspiration that occur in the dry sauna do not occur when most of the body is under water in spas. Although the transfer of heat is more rapid in spas than in saunas, the gradient of heat is less. Water in spas is generally not above 40°C, which is in contrast to the 80°C or 90°C temperature of the Finnish sauna or the 50°C temperature of the steam room or Japanese sauna.

In addition to the problems of hyperthermia the hazard of drowning exists in spas. Infants can accidentally fall into the spa and drown when the spa is not adequately covered; or older children can become entrapped if the powerful outflow suction is not properly shielded. As noted in Table 2, over half of the 104 deaths reported to the CPSC were in children under 12 years of age. Shinaberger et al.[9] in a study of 74 deaths of young children in California spas found that the major factors involved were lack of supervision, access to the spas, neuromotor handicaps, and entrapment by suction.

Syncope is a hazard for adults. Hyperthermia may induce syncope, particularly in persons with heart disease or alcohol or drug ingestion. Even without these factors, staying in the spa too long or allowing the temperature to rise beyond safe limits can cause syncope.

The greater likelihood of narrowed arteriosclerotic blood vessels in the heart and brain of the elderly poses a risk, especially if they are perspiring profusely and, as a result, become dehydrated and have increased blood viscosity. Alcohol or cocaine, medications that affect the heart or the temperature-regulating mechanism or that result in drowsiness are more likely to be present in adults. Trying to sober up or counter a hangover by "sweating it out" can be a perilous activity. Ylikhari et al.[10] noted that in Finland, which has a population of 4.8 million, the consumption of alcohol has been estimated to be a contributing factor in 20 to 25 sauna-related deaths each year. He felt that alcohol intoxication and particularly the hangover phase exposes a person to cardiac arrhythmias and that the sauna may further increase the arrhythmia risk because of enhanced adrenergic activity.

The length of stay in a sauna or spa is important. The heating switch on some electrically heated saunas is set to go off automatically after 30 minutes, and one of the standards (currently under revision) cautions against exceeding 30 minutes. However, Sohar et al.[4] found even 20 minutes to be too long for 4 of his 60 subjects as noted above. The New York City Health Department requires posting a notice in public saunas, steam rooms, and spas with the following warning: "Use . . . should not exceed 15 minutes. Excessive exposure can be harmful to health" (personal communication, November 1987, between the commissioner of the New York City Health Department and the chair of the CPSC). The president of the Institute for Health Promotion Research in Finland who has done research and had an unusually extensive experience with saunas stated that "—even 10 minutes may be too long a sauna exposure for some individuals." In 2 of the 10 fatalities in the author's survey where the time was recorded the time was less than 10 minutes. Studies to determine how long pregnant women can safely remain in a spa found that the length of time varied with the temperature of the water.[11] However, using 38.9°C as a potentially teratogenic temperature, Harvey et al.[11] found that in a hot tub with water at 39°C it took the women 15 to 25 minutes (mean 23.0 minutes) to reach a body temperature of 38.9°C. When the water temperature was 41.1°C it took the women 10 to 30 minutes (mean 18.5 minutes). In a sauna at 81.4°C all 20 women felt sufficient discomfort (dizziness, tingling in hands, rapid pulse, irregular heart beat, and stomach pain) to leave before their temperature rose to 38.9°C. The subjects in the 39°C hot tubs began to leave because of discomfort after 10 minutes; those in the 41.1°C tub left after 5 minutes. Those who elected to remain because they did not experience any discomfort required the above noted times to have their temperatures reach 38.9°C.

Spas, sometimes marketed through discount stores at cut rates are often purchased by families with small children. The combination of inadequately supervised children and the use of cheaper models with substandard covers and poorly shielded suction outlets could result in accidental drowning. Mandatory, rather than voluntary, safety standards for covers and baffles might decrease this hazard. Many of the standards developed by several organizations need to be revised, and for all compliance is now voluntary. Standards have been formulated by Underwriters Laboratories[12] and the National Spa and Pool Institute.[13] The Centers for Disease Control of the US Department of Health and Human Services has also published some recommended health and safety guidelines for public spas and hot tubs.[14] A few state or local jurisdictions have adopted some of these standards (with or without recent revisions) as requirements for public facilities; they may also enforce them. However, most official agencies have taken no action. Moreover, the current mandatory standards of most official agencies cover only public and not private residential facilities. The CPSC has the authority and responsibility to promulgate mandatory standards for both public and private saunas and spas but, as of December 1990, has not done so.

Both the author's survey and the CPSC data suffer from incomplete information: the total population at risk, total number of associated deaths, and the number, type and frequency of use are not known. The sparse response rate to my survey as well as its lack of complete coverage means that many deaths and many risk factors may have been overlooked. A more thorough and complete study is needed, preferably a prospective one, to obtain a more accurate and complete picture of the problem.

Nevertheless, even the limited data presented here suggest that a public health problem exists and that the problem can be corrected through appropriate actions by the CPSC and official health agencies.

Persons with heart disease, hypertension, seizure disorders, diabetes, or significant obesity; persons who have ingested alcohol, narcotic drugs, or medications that can result in drowsiness or interfere with the body's temperature-regulating mechanism; or persons who are over 65 years of age must limit their stays in saunas or spas to 5 or at the most 10 minutes at a time. Similar precautions should be observed by women in the first trimester of pregnancy. Even healthy adults would be well advised not to stay in for more than 10 to 15 minutes at a time.

Automatic controls, limiting air and water temperatures to 80°C for the Finnish Sauna, 50°C for the steam room, and 40°C for the water temperature in the spas should be required as should effective covers for spas and proper baffles or shields for suction outlets. ☐

Acknowledgment

A condensed version of this article was presented on September 10, 1990, at a joint meeting of the Oregon and Washington State Public Health Associations.

References

1. Kauppinen K, Vuori I. Man in the sauna. *Ann Clin Res.* 1986;173–185.
2. Sorri P. The sauna and sauna bathing habits—a psychoanalytical point of view. *Ann Clin Res.* 1988;236–239.
3. Vuori I, Heiddi V, eds. Special issue on sauna. *Ann Clin Res.* 1988;20:215–294.
4. Sohar E, Shoenfeld Y, Ohry A, Cabili S. Effects of exposure to Finnish sauna. *Isr J Med Sci.* 1976;12:1275–1282.
5. Jokinin E, Valinaki I, Antilla K, Seppanen A, Tuominen J; Children in sauna: cardiovascular adjustment. *Pediatrics.* 1990;86:282–288.
6. Kohgali M, Gales JRS. Editors' comments. In: Kohgali M, Hales JRS, eds. *Heat Stroke and Temperature Regulation.* Sydney, Australia: Academic Press (Harcourt-Brace Jovanovich); 1983;XIII–XVI.
7. Edwards MJ. Hyperthermia as a teratogen: a review of experimental studies and their clinical significance. *Teratogenesis Carcinog Mutagen.* 1986;6:563–582.
8. Vaha-Eskeli K, Erkkola R. The sauna and pregnancy. *Ann Clin Res.* 1988;20:279–282.
9. Shinaberger CS, Anderson CL, Kraus JF. Young children who drown in hot tubs, spas, and whirlpools in California: A 26-year survey. *Am J Public Health.* 1990;80:613–614.
10. Ylikhari R, Heikkonin E, Soukas A. The sauna and alcohol. *Ann Clin Res.* 1988;20:287–291.
11. Harvey SMA, McRorie MM, Smith DW. Suggested limits to the use of the hot tub and sauna by pregnant women. *Can Med Assoc J.* 125:50–53.
12. Underwriters Laboratories. *Standard for Electric Sauna Heating Equipment, UL # 875, 4th Rev & UL Standard # 1563 for Electric Hot Tubs, Spas and Associated Equipment.* 2nd ed. Santa Clara, Calif.; 1989.
13. National Spa and Pool Institute (NSPI). *Standards for Permanently Installed Public Spas, NSPI 2, 1989; Standards for Permanently Installed Residential Spas, NSPI 3, 1989; Standards for Portable Spas, NSPI 6, 1989.* (These NSPI Standards were under revision as of Dec. 1990) Alexandria, Va: NSPI.
14. *Suggested Health and Safety Guidelines for Public Spas & Hot Tubs.* 2nd ed. Atlanta, Ga: Centers for Disease Control; 1985.

Serum Total Cholesterol: HDL Cholesterol Ratios in US White and Black Adults by Selected Demographic and Socioeconomic Variables (HANES II)

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ABSTRACT

Background: Framingham Study findings suggest that total cholesterol (TC):High density lipoprotein cholesterol (HDL-C) ratio is a useful summary of the joint contribution of TC and HDL-C to coronary heart disease (CHD) risk. Information on the distribution of TC:HDL-C in the US population is limited to selected populations and the relationship of the ratio distribution and its correlates has received little attention.

Method: TC/HDL-C ratios were examined in a representative sample of the United States adult population ages 20 to 74 years, between February 1976 and February 1980 during NHANES II, using stratification and multivariate regression analyses.

Results: Age-adjusted mean ratios were higher in men compared with women and were higher in Whites compared with Blacks. White men had the highest TC/HDL-C mean ratios. These relationships remained after stratification by age, education, body mass index, alcohol use, cigarette smoking, and physical activity. Using multivariate analyses, the ratios were positively related to BMI, age, and smoking; and negatively related to female sex, alcohol use, being Black, and physical activity.

Conclusions: Using a ratio reference point of greater than or equal to 4.5 from the Framingham study, at least an estimated 44 million persons ages 25 to 74 years in the US were found to be at higher risk of developing coronary heart disease. (*Am J Public Health*. 1991;81:1038–1043)

Introduction

The various cholesterol-carrying lipoproteins are independent predictors of coronary heart disease (CHD) risk. Plasma total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) levels are positively correlated with CHD, whereas high density lipoprotein cholesterol (HDL-C) levels are inversely related.[1] However, TC or LDL-C and HDL-C are poorly correlated with each other so that knowledge of both may be important to predict CHD risk than does either of them alone.

Various ratios combining TC or LDL-C and HDL-C have been introduced. Framingham Study findings suggest that TC:HDL-C ratio is a useful summary of the joint contribution of TC and HDL-C to CHD risk.[1-3]

Other summary measures, using LDL-C, may need calculations or special laboratory methods. We have chosen to report the TC:HDL-C to allow comparison with the Framingham data, since it is the main source of information on the relationship of a summary measure of TC and HDL-C with CHD mortality. This ratio has the advantage of being available to the physician and is easier to comprehend at the clinical level.

Information on the distribution of TC:HDL-C (or its inverse) in the United States population is limited to selected populations. Both the Framingham Study[1,3] and the Lipid Research Clinics (LRC) Prevalence Study[4] were not planned to be representative of the US population. Furthermore, the relationship of the ratio distribution and its correlates has received little attention.

The second National Health and Nutrition Examination Survey (NHANES II),[5,6] measured TC and HDL-C levels in a probability sample of the civilian, noninstitutionalized population of the United States and thus provides information on the distribution of the TC:HDL-C ratio for categories of selected potential correlates, namely age, sex, race, education, body mass index (BMI), alcohol intake, smoking, and physical activity.

Methods

The NHANES II, conducted between February 1976 and February 1980 by the National Center for Health Statistics (NCHS), was a national cross-sectional probability survey of the civilian noninstitutionalized population of the United States aged 6 months to 74 years including Alaska and Hawaii.[5,6] It consisted of household interviews, as well as physical examination and interviews in mobile examination centers. The household interview collected socioeconomic

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TABLE 1—NHANES Study population: Numbers and response rates by sex and race

		Men		Women	
	Total*	White	Black	White	Black
Total sample	17058	7059	852	8044	1103
Interviewed	15080	6170	766	7146	998
Response rate (%)	88.4	87.4	89.9	88.8	90.5
Examined	11637	4483	607	5418	729
Response rate (%)	77.2	79.1	79.2	75.8	73.0
HDL determinations	9625	4019	462	4563	581
Response rate (%)	82.7	82.3	76.1	84.2	79.7
Overall response Rate (%)	56.4	56.9	54.2	56.7	52.7

*Includes only Whites and Blacks aged 20–74 years.

and demographic information on the sample person and the family and a medical history questionnaire. The medical examination was conducted according to a standardized protocol and included laboratory tests on whole blood and sera. For each person examined, dietary data including alcohol consumption were obtained by means of a 24-hour recall and a three-month food frequency questionnaire administered by trained dietary interviewers.

The three sources of nonresponse and response rates by sex and race are shown in Table 1. An analysis of selected health variables of persons who were examined versus those who were not examined (but interviewed) indicated no substantial bias due to nonresponse.[7] An examination of the characteristics of persons with and without HDL-C determinations such as age, education, BMI, alcohol use, smoking, and physical activity did not detect differences that would suggest a bias in the results. Adjustments have been made according to the usual NCHS methods so that the results reported here reflect a nationally representative sample.[5]

Determination of Serum Lipids

Chemical analyses were provided by the LRC Laboratory at the George Washington University, and the editing and data processing were provided by the LRC Central Processing Unit, Department of Biostatistics, at the University of North Carolina. The laboratory techniques have been described elsewhere.[5,8]

Definitions

Educational level: Measured by the highest grade attended in a public or a private school.

Body mass index (BMI, Quetelet index): Weight (Kg)/height (m)}**2.

Frequency of alcohol consumption: Participants were asked about the usual consumption of alcoholic beverages during the three months that preceded the interview. Consumption was classified as follows: none (never), less than once/week (seldom), 1 to 6 times/week (weekly), or 1 or more times/day (daily). The data for all types of alcohol—beer, wine, and liquor—were compared. The greatest frequency of reported alcohol use was calculated as a composite measure of habitual (chronic) frequency of alcohol use of any kind.

Smoking: Interviewers were asked: 1) whether they had ever smoked at least 100 cigarettes during their entire life; 2) whether they currently smoked; and 3) the daily number of cigarettes smoked. Individuals who responded positively to questions #1 and #2 were defined as "current smokers." Those who responded positively to question #1 but negatively to question #2 were defined as "past smokers," and those who responded negatively to question #1 were defined as "nonsmokers." For current smokers, the data were dichotomized into those who smoked fewer than 21 cigarettes per day, and those who smoked 21 or more cigarettes per day.

Physical activity: Interviewees were asked: 1) whether they were getting "much exercise," "moderate exercise," or "little or no exercise" for recreation; 2) whether in their usual day, aside from recreation, they were "very active," "moderately active," or "quite inactive." The higher category of activity, at any time was then calculated as a composite of activity of any kind.

Statistical Methods

All statistical analyses incorporated both the weights and the complex sample design of the NHANES II. Age-specific and age-adjusted mean HDL-C levels of men and women by race and by selected correlates were estimated by use of the programs SURREGR[9] and GENCAT.[10,11] The age-adjusted means were standardized by the direct method to the 1980 US census population. The first level of analysis consisted of testing the equality in age-specific mean and the age-adjusted mean ratio by selected correlates for the four sex-race groups. Hypotheses of no difference in mean ratio were tested at the 0.05 level of significance, using a chi-square statistic with one degree of freedom. For this report the use of the word "significant" implies "statistically significant."

Second, Pearson correlation coefficients were estimated to investigate the relationship between the ratio and continuous variables such as age, years of education, and the number of cigarettes currently smoked. Correlation coefficients and standard errors were estimated by use of the program OSIRIS.[12] Since the sampling distribution of the correlation coefficients is not normal, Fisher's Z transformation of the correlation coefficients was used to test the hypothesis of zero correlation.

Third, using the SURREGR program, weighted least squares multiple regression analyses,[9] with ratio as the dependent variable, were applied to control for potential confounding. All correlates were included simultaneously in the equation.

Results

The mean ratios and selected percentiles by age, sex, and race are presented in Table 2. The ratios were directly related to age ($P < 0.001$) with Pearson correlation coefficients of 0.235 for White men, 0.207 for Black men, 0.301 for White women, and 0.247 for Black women. White men had the highest mean ratios, consistently increasing from 4.11 at ages 20 to 24 to a peak of 5.55 at ages 45 to 54, and declining thereafter.

The ratios for White men were consistently and significantly higher than those of White women. The age-adjusted sex difference for Whites was 0.77 (18 percent higher for males). Black men had higher ratios than Black women at ages 25 to 54, but Black women had higher ratios in the youngest age group 20 to 24 and at age 55 and above. Blacks had consistently lower ratios than Whites, for both men and women. All differences for men were significant.

TABLE 2—Ratio of Total Cholesterol to HDL Cholesterol (mg/dl) of Adults Ages 20–74 Years by Sex, Race, and Age, with Means, Age-Adjusted Means, and Selected Percentiles, United States 1976–80

	Examined Persons	Estimated Population in Thousands	Selected Percentiles									
			Mean	5th	10th	15th	25th	50th	75th	85th	90th	95th
MEN												
Whites												
20–74	4,019	55 806	5.06	2.79	3.17	3.43	3.88	4.81	5.96	6.72	7.29	8.13
Age-adjusted mean ratio			5.05									
20–24	494	8 052	4.11	2.39	2.72	2.95	3.25	3.98	4.75	5.31	5.07	6.51
25–34	751	13 864	4.75	2.58	3.05	3.32	3.74	4.51	5.53	6.22	6.72	7.57
35–44	545	9 808	5.31	3.14	3.41	3.62	4.06	4.97	6.26	6.97	7.79	8.21
45–54	506	9 865	5.55	3.14	3.50	3.86	4.35	5.39	6.49	7.24	7.78	8.79
55–64	865	8 642	5.47	3.08	3.47	3.80	4.17	5.19	6.51	7.25	7.82	8.54
65–74	858	5 576	5.30	2.86	3.32	3.55	4.03	5.11	6.24	6.84	7.39	8.25
Blacks												
20–74	462	6 102	4.28	2.24	2.59	2.77	3.08	4.00	5.11	5.69	6.22	7.36
Age-adjusted mean ratio			4.33									
20–24	65	1 043	3.43	+	2.24	2.44	2.64	3.22	4.20	4.41	4.70	+
25–34	112	1 546	4.16	2.37	2.59	2.74	3.06	3.91	5.04	5.64	6.06	7.00
35–44	54	1 112	4.69	+	2.91	3.34	3.53	4.45	5.28	6.07	6.75	+
45–54	47	1 044	4.75	+	+	2.83	3.54	4.17	5.44	6.09	+	+
55–64	92	801	4.50	+	2.79	3.00	3.38	4.28	5.47	6.30	6.69	+
65–74	92	555	4.49	+	2.67	2.84	3.32	4.13	5.18	5.69	6.64	+
WOMEN												
Whites												
20–74	4,563	60 785	4.30	2.42	2.68	2.88	3.18	3.98	5.04	5.79	6.30	7.24
Age-adjusted mean ratio			4.28									
20–24	542	8 408	3.66	2.23	2.43	2.59	2.90	3.42	4.20	4.64	5.14	5.72
25–34	875	14 494	3.90	2.40	2.58	2.74	2.98	3.63	4.51	5.07	5.58	6.81
35–44	626	10 584	4.20	2.45	2.66	2.86	3.19	3.98	4.97	5.56	6.06	6.74
45–54	546	10 369	4.57	2.45	2.80	3.00	3.33	4.35	5.47	6.05	6.60	7.60
55–64	973	9 601	4.81	2.63	2.91	3.12	3.57	4.52	5.67	6.56	7.18	8.17
65–74	1,001	7 329	5.00	2.88	3.16	3.36	3.83	4.75	5.89	6.73	7.16	8.03
Blacks												
20–74	581	7 579	4.06	2.30	2.51	2.69	2.98	3.70	4.70	5.60	6.26	7.05
Age-adjusted mean ratio			4.10									
20–24	77	1 304	3.45	+	2.39	2.57	2.67	3.22	4.13	4.68	4.95	+
25–34	122	1 953	3.76	2.22	2.38	2.52	2.84	3.41	4.47	5.45	5.86	6.47
35–44	84	1 415	4.12	+	2.45	2.69	2.96	3.87	4.68	5.39	6.52	+
45–54	81	1 215	4.35	+	3.00	3.07	3.19	3.96	5.16	6.42	6.64	+
55–64	99	959	4.78	+	2.60	2.96	3.11	4.21	5.95	7.19	7.64	+
65–74	118	733	4.50	2.52	2.97	3.23	3.59	4.32	5.15	5.78	5.81	7.07

[1] Age-adjusted by direct method to the 1980 U.S. Census Population.

+Sample size is inadequate to produce a reliable estimate.

TC:HDL-C by Categories of Correlates

Education: In Whites, there was a slight initial increase in the ratios from the first to the second category of education, and a consistent decline thereafter. However, for Black men there was a decrease in the ratio from the first to the second level of education, and a consistent increase with higher education. There was no clear trend in the ratio for Black women. The correlation coefficients were negative in all four sex-race groups, suggesting an inverse relationship between education and the ratio (data available on request from authors).

BMI: The ratios increased uniformly in all four sex-race groups with increasing BMI (Table 3). The relationships of the ratio with BMI were stronger in women than in men, and in Blacks than in Whites.

Alcohol: The age-adjusted mean of Whites of both sexes and of Black women decreased consistently with increased frequency of alcohol consumption (Table 4). The ratio of Black men was highest for those who seldom consumed alcohol and decreased consistently thereafter. For each of the four sex-race groups except for Black men there were lower ratios with increasing reported quantities of alcohol consumed over the last 24 hours. The Pearson correlation coefficients were stronger in men than in women and in Whites than in Blacks.

Smoking: In all groups with the exception of Black men, nonsmokers had lower ratios than smokers (Table 5). However, Black men who never smoked and past smokers had higher ratios than light smokers. Among the smokers, the heavier the smoking the higher the ratio in all groups with the exception of Black females, where the number of cases was too small to produce a reliable estimate.

Physical activity: White men and women, and Black men and women had the lowest ratios with highest level of reported physical activity (Table 6).

TC:HDL-C Ratio in the Four Sex-Ratio Groups after Stratification for Correlates

Sex and race patterns remained basically similar to the findings in the basic data even after controlling for correlates: Of the four sex-race groups, White men consistently had the highest ratios; and Black women had the lowest ratios after stratification for BMI and alcohol consumption (data available on request from authors).

Multivariate Analyses

In the multivariate model with all correlates (Table 7), BMI, age, and current smoking were positively and independently related to the ratio. Being female, frequent alcohol use, being Black, and physical activity were negatively and independently related to the ratio. In this model, education was not strongly related to the ratio. Similar results were obtained when the natural log of the ratio was used as a dependent variable.

Discussion

The present report provides estimates of the distribution of the TC:HDL-C ratio and the relationship to selected correlates in a representative sample of the United States population. The ratios were found to increase with age in men and women of both races. The ratios were consistently higher for men compared to women for the two races; and Whites had higher ratios compared to Blacks. These relationships remained even after stratification for categories of correlates.

The age and sex trends of the TC:HDL-C ratios were similar to those seen in the Framingham study which, however, found higher values. The socioeconomic status (SES) of the Framingham population is higher than that of the general US population. However, since higher SES was found to be related to lower ratios in the present study, SES does not account for the differences observed in the two studies. Laboratory and other methodological differences in lipids and lipoprotein determinations could account for some of the differences. A bias in the determination of lipid levels in NHANES II seems to be unlikely. All TC and HDL-C determinations were performed using a standardized procedure, without knowing the participants demographic characteristics and the quality of the laboratory determinations were routinely evaluated.

In the univariate analyses the ratio was negatively related to years of education in the four sex-race groups but when other correlates were considered in a multivariate model, the ratio was only weakly positive and weakly related to education.

TABLE 3—Age-Adjusted Mean Ratio of Total Cholesterol to HDL Cholesterol of Adults, Ages 20–74 Years, by Race, Sex, and Quartiles of Body Mass Index (BMI), and Pearson Correlation Coefficient, United States, 1976–80

Men Quartiles of BMI	*White*	*Black*
Less than 22.7	4.45	3.59
22.7–24.9	4.86	4.07
25.0–27.5	5.25	4.65
27.6 or more	5.67	5.10
Pearson Correlation	0.302*	0.358*
Women Quartiles of BMI	*White*	*Black*
Less than 21.2	3.82	3.25
21.2–23.7	4.06	3.52
23.8–27.4	4.37	3.98
27.5 or more	4.99	4.60
Pearson Correlation	0.313*	0.367*

Age-adjusted by direct method to the 1980 US Census population.
Body mass index quartiles were determined from sex-specific BMI distribution.
*$P < 0.001$

TABLE 4—Age-Adjusted Mean Ratio of Total Cholesterol to HDL Cholesterol of Adults, Ages 20–74 Years, by Race, Sex, and Frequency of Consumption of Alcoholic Beverages, United States, 1976–80

Frequency of Consumption	White Men	Black Men	White Women	Black Women
Never	5.42	4.44	4.51	4.30
Seldom	5.20	4.65	4.32	4.19
Weekly	4.92	4.19	4.05	3.76
Daily	4.74	3.77	3.63	3.53

Age-adjusted by direct method to the 1980 US Census population.

Frequency of consumption of alcoholic beverage was determined by taking the maximum reported consumption of either beer, wine, or liquor from the food frequency questionnaire.

TABLE 5—Age-Adjusted Mean Ratio of Total Cholesterol to HDL Cholesterol of Adults, Ages 20–74 Years, by Race, Sex, and Smoking Status, and Pearson Correlation Coefficient, United States, 1976–80

Smoking Status	White Men	Black Men	White Women	Black Women
Nonsmokers	4.97	4.46	4.19	4.03
Past smokers	4.99	4.51	4.12	4.21
Smokers				
1–20 cigarettes/day	5.05	4.01	4.45	4.17
21 cigarettes or more/day	5.38	4.54	4.53	—
Pearson Correlation	0.141*	—	0.049	—

Age-adjusted by direct method to the 1980 US Census population.

A past-smoker is defined as a person who had smoked at least 100 cigarettes but does not currently smoke; a nonsmoker did not smoke 100 cigarettes in his lifetime and does not currently smoke.

*$P < 0.001$.

—Sample size is inadequate to produce a reliable estimate.

Some limitations on the use and interpretation of the ratio may be advised. It is important to emphasize that the TC:HDL-C can change as a function of either TC or HDL-C. Furthermore, the relationship to correlates is complicated by the fact that at any given ratio level a number of correlates can influence infinite combinations of TC and HDL-C and be responsible for the final outcome. Some correlates such as age will have their effect via TC since there is no strong relationship between age and HDL-C. Conversely, alcohol consumption, for

TABLE 6—Age-Adjusted Mean Ratio of Total Cholesterol to HDL Cholesterol of Adults, Ages 20–74 Years, by Race, Sex, and Physical Activity, United States, 1976–80

Physical Activity Level	White Men	Black Men	White Women	Black Women
Quite inactive	5.31	4.31	4.46	4.80
Moderately active	5.12	4.49	4.28	4.06
Very active	4.96	4.12	4.23	3.89

Age-adjusted by direct method to the 1980 US Census population.

TABLE 7—Multiple Linear Regression Model with Ratio of Total Cholesterol to HDL-Cholesterol as a Dependent Including all Correlates

Overall R^2: 0.212 Intercept 1.969	B[a]	partial R^2 (%)
BMI	0.089[b]	9.16
Age (years)	0.022[b]	4.16
Sex (women vs men)	−0.773[b]	4.34
Current smoker	0.409[b]	1.23
Alcohol use (never or seldom vs others)	−0.430[b]	1.13
Race (Blacks vs Whites)	−0.575[b]	1.10
Physical activity (very active or moderate vs inactive)	−0.089[c]	0.07
Education (years)	0.002	0.00

[a] Estimated regression coefficient.
[b] $P < 0.001$.
[c] $P < 0.05$.

example, will predominantly affect HDL-C. BMI on the other hand is known to influence both components of the ratio, and indeed proved to be the most important predictor of TC:HDL-C in the multivariate analyses. Several publications have investigated the relationships of TC or HDL-C to demographic and socioeconomic variables in the US.[5,13–15] When assessing the distribution of the ratio and its correlates, these factors must be taken into consideration.

In the Framingham Study, the TC:HDL-C ratio was found to be an excellent predictor of CHD risk. Men and women who did not develop CHD within the years of follow-up had an average ratio of 5.2. For those who did develop CHD over the same period, the average ratio for men was 5.8 and for women 5.3. The American Heart Association guidelines suggest that to consider all those at or above ratios of 4.5 be considered at higher risk of CHD[1,16] as TC:HDL-C ratio of 4.4 corresponds to total cholesterol level of 200 mg/dl and HDL-C of 45 mg/dl. Using this single reference ratio, Castelli[1] estimated that 50 percent of the women and 75 percent of the men in the United States are at risk of CHD. Better estimates may be obtained using the NHANES II data (Table 2). Thus, 50 percent of the White males and at least 25 percent of the Black males ages 25 and above have ratios at or above 4.5 and would be considered at increased risk of CHD. This corresponds to population estimates of 23,877,500 White males and 1,264,500 Black males. Similarly, at least 25 percent of women, of both races, ages 25 and above would be at higher risk, corresponding to population estimates of 17,326,750 White women and 1,568,750 Black women, respectively. Thus, there are about 44 million White and Black persons in the US ages 25 to 74 who may be at higher risk of developing CHD based on a TC:HDL-C ratio of at or above 4.5. However, it must be emphasized that there is a need to describe the shape of the curve of the TC/HDL-C levels and CHD risk before selecting a reference point to obtain a reliable estimate of the population at higher risk.

The use of TC:HDL-C to predict CHD may be an oversimplification. In only one study in the US, the Framingham Study, has the predictive power of the ratio been determined. Furthermore, it is unclear whether the ratio predicts CHD across all levels of TC.[16,17] □

References

1. Castelli WP. Epidemiology of coronary heart disease: The Framingham Study. *Am J Med.* 1984;76(2A):4–12.
2. Kannel W, Castelli WP. Is the serum total cholesterol an anachronism? (editorial) *Lancet.* 1979;2:950–951.
3. Castelli WP, Abbott RD, McNamara PM. Summary estimates of cholesterol used to predict coronary heart disease. *Circulation.* 1983;67:730–734.
4. Green MS, Heiss G, Rifkind BM, Cooper GR, Williams OD, Tyroler HA. The ratio of plasma high density lipoprotein cholesterol to total and low density lipoprotein cholesterol: Age-related changes and race and sex differences in selected North American populations. The Lipid Research Clinics Program Prevalence Study. *Circulation.* 1985;72:93–104.
5. National Center for Health Statistics. Total serum cholesterol levels of adults 20–74 years of age, United States, 1976–1980. NCHS, DHHS Pub. No. (PHS) 86-1686.
6. National Center for Health Statistics, Engel A, Massey J, Maurer K. Plan and operation of the second National Health and Nutrition Examination Survey 1976–1980. Vital and Health Statistics, Series I No. 15, DHEW Pub. (PHS) 81-1317. Health Research, Statistics, and Technology. Washington, DC: Govt Printing Office.
7. Forthofer RN. Investigations of nonresponse bias in NHANES II. *Am J Epidemiol.* 1983;117:507–515.
8. National Institutes of Health. Manual of laboratory operations Volume 1, Lipid Research Clinics Program, Lipid and lipoprotein analysis. DHEW Pub. No. (NIH) 75-628, 1974.
9. Holt M. SURREGR—standard errors of regression coefficient from sample survey data. Research Triangle Park, NC: Research Triangle Institute, May 1977; Revised April 1982.
10. Koch GG, Freedman DH, Freedman JL. Strategies in the multivariate analysis of data from complex surveys. *Int Stat Rev.* 1975;43(1):59–78.
11. Landis JR, Stanish WM, Freedman JL, Koch GG. A computer program for the generalized chi-square analysis of categorical data using weighted least squares (GENCAT) *Comput Programs Biomed.* 1976;6:196–231.
12. Survey Center Computer Support Group, OSIRIS IV User's Manual. Ann Arbor, Mich: Institute for Social Research, 1979.
13. US Department of Health and Human Services, Public Health Service, National Institutes of Health. The Lipid Research Clinics Population Studies Data Book. NIH Pub. No. 80-1527, July 1980.
14. Lipid Research Clinics Program: Plasma lipid distributions in selected North American populations: The Lipid Research Clinics Program Prevalence Study. *Circulation.* 1979;60:427–439.
15. Linn S, Fulwood R, Rifkind B, Carroll M, Muesing R, Johnson C. High density lipo-

protein levels among US adults by selected demographic and socioeconomic variables. The Second National Health and Examination Survey 1976–1980. *Am J Epidemiol.* 1989;129:281–294.

16. Grundy SM, Greenland P, Herd A, Huebsch JA, Jones RJ, Mitchell JH, Schlant RC. Position Statement, American Heart Association. Cardiovascular and risk factor evaluation of healthy American adults. *Circulation.* 1987;75:1339A–1362A.
17. Consensus Development Conference Statement on Lowering blood cholesterol to prevent heart disease. *JAMA.* 1985;253:2080–2086.

Late Breaker Session on Injury Control Invites Abstracts

The Injury Control and Emergency Health Services special primary interest group of the American Public Health Association has announced it will again feature a "late breaker" session during the upcoming 119th APHA annual meeting in Atlanta. The session will be held on Tuesday, November 12, 8:30 AM–10:00 AM, and will feature work completed within the last few months—after the deadline for consideration in the regular symposia of the APHA annual meeting.

Abstracts of 250 words or fewer will be accepted by the Injury Control SPIG until September 10, 1991. Please send the abstract, title of the paper, author's name, address, and telephone number to: Richard Waxweiler, Division of Injury Control, Centers for Disease Control, Mail Stop F-36, Atlanta, GA 30333. Tel: 404/488-4695.

Public Health Briefs

Physicians Promoting Bicycle Helmets for Children: A Randomized Trial

Robert Cushman, MD, FRCP, William James, MD, FRCP, and Helen Waclawik, BScN, RN

ABSTRACT

Head injury is the leading cause of death and serious morbidity in bicycle accidents. There is good evidence to recommend helmets, yet few children wear them. We evaluated helmet promotion in a randomized trial targeting children presenting to primary care settings for routine ambulatory care. The intervention consisted of physician counseling and take-home pamphlets. The study involved 339 families, 167 in the intervention group and 172 in the control group. In a follow-up telephone call, 2 to 3 weeks later, only 7.2% of the intervention group had purchased helmets, compared with 7.0% of the control group (χ^2 = 0.0056, P = .94). During the latter half of the study, bicycle safety received considerable media attention in Ottawa, and the provincial medical society sponsored a $5 discount campaign. Therefore both groups were subject to community "co-intervention." Nonetheless, we were surprised that physician counseling made no additional impact. Our results and the success of certain community programs suggest that physicians interested in helmet promotion would do better to participate in the design and implementation of multidisciplinary campaigns. (*Am J Public Health.* 1991;81:1044-1046)

Introduction

Head injuries account for 70% to 80% of deaths and serious morbidity in bicycle-related injuries.[1-7] Children and young adults are at greatest risk, accounting for more than 50% of deaths, and cycling is well recognized as a leading cause of head injury in childhood.[8-13] There is ample evidence to recommend the use of helmets, yet relatively few cyclists wear them.[14-22] Surveys in Arizona and Ontario found helmet use was less than 2% among elementary and secondary school children, compared with rates of 10% to 20% in the adult population.[10,22]

This study was designed to evaluate the effectiveness of promoting the use of bicycle helmets during a routine primary care visit.

Methods

Study Design

We targeted families with one or more cyclists ages 5 to 18, presenting to their pediatrician's or family doctor's office for a routine ambulatory visit. Physicians exercised discretion and excluded families presenting for emergency care or other special circumstances, where helmet promotion would have been inappropriate. All participants received an envelope containing a brief one-page survey and a consent form. Parents were asked to list the cyclists by age and to provide information about bicycles and helmet ownership and use. Randomly flagged envelopes indicated to the physician which families were to receive the intervention. The control group received only the usual care.

The Intervention

The intervention consisted of helmet promotion counseling and take-home pamphlets. During an individualized orientation session, physicians were provided with a small plasticized cue card outlining facts about bike accidents and head injuries (Table 1). Physicians were also encouraged to use the pamphlets as a focus for discussion and to express their personal concerns about the dangers of riding without protective headgear.

Outcome Measure

At 2 to 3 weeks' postintervention, a research assistant made a follow-up phone call to tabulate helmet purchases. Positive respondents were also questioned about cost, brand, and place of purchase. The answers allowed us to assess the validity of the "yes" responses. Because helmet owners often ride without headgear, additional questions addressed helmet use.[23,24]

Statistical Analysis

We estimated a helmet purchase rate of 5% to 10% in the control group, and for our efforts to be worthwhile, sought a rate 2 to 3 times higher in the intervention group. Based on our calculations, using a

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This paper was submitted to the journal September 13, 1990, and accepted with revisions March 6, 1991.

Editor's Note: See related editorial on p. 972.

TABLE 1—Helmet Promotion Cue Card Contents

1. Last summer over 100 children with bike related injuries were admitted to the children's hospital.
2. Every year more than 600 children in the U.S.A. die from bike accidents.
3. Most serious injuries and deaths result from head injury.
4. Bike accidents are the leading cause of head injury in childhood.

Bike helmets can reduce the number and severity of head injuries. We consider helmets to be standard equipment and we are encouraging all our patients who cycle to purchase and use them.

one-tailed α of .05 and a β of .1 to .2, we determined that a sample size of 300 would provide adequate power. It would allow us to detect an absolute increase of 10% in the helmet purchase rate, from 5% to 15% at the low end of the scale and from 10% to 20% at the high end. The χ^2 was used to compare the number of helmets purchased in the two groups. Baseline characteristics were assessed using χ^2 or t tests where appropriate.

Results

There were 167 families in the intervention group and 172 in the control group. A total of 288 children were allotted to each group. Small differences between the groups were not statistically significant (Table 2).

At follow-up, only 12 (7.2%) of the 167 families in the intervention group had purchased helmets, compared with 12 (7.0%) of the 172 families in the control group. The 0.2% difference in favor of the intervention group was neither clinically nor statistically significant ($\chi^2 = 0.0056$, $P = .94$). Calculation of the 95% confidence interval on this difference gives a margin of error of ±5.5%, meaning that there is only a 2.5% chance that the intervention might have increased the purchase of helmets by 5.7% (0.2 ± 5.5) (Table 3).

TABLE 2—Subject Baseline Characteristics

Characteristic	Intervention (n = 167)	Control (n = 172)	Total (N = 339)
No. of children	288	288	576
Age			
Mean (years)	9.62	9.30	9.44
SD	3.43	3.14	3.29
Range	5–17	5–17	5–17
Families owning one or more helmets for cycling (%)	24.6	27.9	26.3

TABLE 3—Helmet Purchases by Family

Group	Purchased Helmet	Did Not Purchase Helmet
Intervention	12	155
Control	12	160

Discussion

While the intervention proved disappointing, we were a little surprised by the control group results. During the latter half of the study (summer 1989) helmet promotion received considerable media attention in Ottawa. The Ontario Medical Association, in conjunction with a local department store, offered a $5 discount on helmets with coupons displayed on posters in waiting rooms. Therefore, community "co-intervention" may have influenced the study. We also considered possible "contamination," whereby clinicians inadvertently offered the intervention to the control group. Physicians were briefed about randomization and the two arms of the study. Only the flagged envelopes contained the pamphlets, serving as the cue for counseling, and when we addressed this issue upon completion of the study, participants did not feel contamination was a problem.

The more important question is why helmet promotion produced no additional effect in the intervention group. We considered three possible problems: low-quality intervention, high rates of helmet use, and price. The intervention was deliberately designed for busy office practice. Counseling sessions were brief, and the material provided a framework for physicians to deliver the message in their own style. Physicians were not "hand-picked" for interest in the subject, nor for counseling skills. However, they were volunteers who received a personalized formal orientation, as well as informal monitoring, from the research assistant.

Approximately 26% of families owned at least one helmet at study entry. On a per capita basis this probably compares with our emergency room finding, 13% in children following a bike injury, and should not have limited the impact of the intervention.[24] Finally, Ottawa is a relatively affluent community with 33% of households reporting an income of more than $50 000 in 1985. Price was identified in our follow-up as a minor deterrent compared with lack of interest and peer pressure.[25]

In a similar study conducted in an emergency room following bike injuries, our helmet promotion efforts were also ineffective.[26] Our experience contains a message of caution, as well as a challenge. Helmet promotion is now encouraged by medical societies. While such interventions may have an impact over time, we suggest the medical community seek additional strategies.[27] There is evidence from Australia, Canada, and the United States that community programs involving the media, schools, and youth groups are effective.[9,28,29] Physicians can play an active role in teaching parents and children that bikes are not just another toy and that helmets can go a long way in reducing the risks of head injury. However, helmets for children are still fairly innovative, and the promotional challenges suggest physicians would be more effective by participating in the design and implementation of multidisciplinary community campaigns. ☐

Acknowledgments

This project was made possible by an emergency health services research grant (No. 00988N) from the Ontario Ministry of Health. We would like to thank all participating physicians and Christine Jenkins of the Ottawa-Carleton Safety Council.

The promotional material used in this study is as follows:

1. "Be bike smart." The Easter Seal Society, 24 Ferrand Drive Don Mills, Ontario, Canada M3C 3N2.
2. "Sprocketman talks road sense." State of North Carolina Department of Transportation, PO Box 25201, Raleigh, NC 27611.

3. "A consumer's guide to bicycle helmets." Washington Area Bicyclist Association, 1015 31st Street NW, 3rd floor, Washington, DC 20007.

References

1. Friede AM, Azzara CV, Gallagher SS, Guyer B. The epidemiology of injuries to bicycle riders. *Pediatr Clin North Am.* 1985;32:141–151.
2. Consumer Product Safety Commission. *NEISS Data Highlights.* 6, No. 3. Washington, DC: Consumer Product Safety Commission; 1982.
3. National Safety Council. *Accident Facts.* Chicago, Ill; 1982:45–91.
4. Lane JC. *Helmets for Child Bicyclists: Some Biomedical Considerations.* Canberra City, Australia: Federal Office of Road Safety; 1986. Report No. CR47.
5. Reynolds R, Cohen D. Children, bicycles and head injuries. *Med J Aust.* 1987; 146:615.
6. Guichon DMP, Myles ST. Bicycle injuries: one-year sample in Calgary. *J Trauma.* 1975;15:504–506.
7. Weiss BD. Childhood bicycle injuries, What can we do? *Am J Dis Child.* 1987;141:135–136.
8. Ontario Medical Association: *Bicycle Accident Fact Sheet.* Toronto; 1989.
9. Brown B, Farley C. The pertinence of promoting the use of bicycle helmets for 8 to 12 year old children. *Chronic Dis Can.* 1989; 10:92–95.
10. Cushman RA, Down J, MacMillan N, Waclawik H. Bicycle helmet use in Ottawa. *Can Fam Physician.* 1990;36:697–700.
11. Lapner M, Leslie PI. Bicycle injuries in children. *Can Med Assoc J.* 1981; 125:132.
12. Armson CJ, Pollard CW. Child cyclist injuries: a prospective study. *Med J Aust.* 1986;144:144–146.
13. Kraus JF, Fife D, Conroy C. Incidence, severity, and outcomes of brain injuries involving bicycles. *Am J Public Health.* 1987;77:76–78.
14. McDermott FT, Klug GL. Head injury predominance: pedal-cyclists vs motor-cyclists. *Med J Aust.* 1985;143:232–234.
15. Waters EA. Should pedal cyclists wear helmets? A comparison of head injuries sustained by pedal cyclists and motorcyclists in road traffic accidents. *Injury.* 1986; 17:372–375.
16. Thompson RS, Rivara FP, Thompson DC. A case-control study of the effectiveness of bicycle safety helmets. *N Engl J Med.* 1989;320:1361–1367.
17. Dorsch MM, Woodward AJ, Somers RL. Do bicycle safety helmets reduce severity of head injury in real crashes? *Accid Anal Prev.* 1987;19:183–190.
18. Bishop PJ, Briard BD. Impact performance of bicycle helmets. *Can J Appl Sport Sci.* 1984;9:94–101.
19. Worrell J. Head injury in pedal cyclists: how much will protection help? *Injury.* 1987;18:5–6.
20. Simpson AH, Unwin PS, Nelson IW. Head injuries, helmets, cycle lanes, and cyclists. *Br Med J.* 1988;296:1161–1162.
21. Kiburz D, Jacobs R, Reckling F, Mason J. Bicycle accidents and injuries among adult cyclists. *Am J Sports Med.* 1986;14:416–419.
22. Weiss BD. Bicycle helmet use by children. *Pediatrics.* 1986;77:677–679.
23. Wasserman RC, Waller JS, Monty MJ, Emery AB, Robinson DR. Bicyclists, helmets, and head injuries: a rider-based study of helmet use and effectiveness. *Am J Public Health.* 1988;78:1220–1221.
24. Cushman R, Down J, MacMillan N, Waclawik H. Bicycle-related injuries: a survey in a pediatric emergency department. *Can Med Assoc J.* 1989;143:108–112.
25. Regional Municipality of Ottawa-Carleton: 1986 Census.
26. Cushman R, Down J, MacMillan N, Waclawik H. Helmet promotion in the emergency room following a bike injury: a randomized trial. *Pediatrics.* In press.
27. Last J. *Maxcy-Rosenau Public Health and Preventive Medicine.* 12th ed.:1118–1120.
28. Road Traffic Authority Australia. *An Exploratory Study of High School Student's Reactions to Bicycle Helmets*; April 1986. *Trends in Helmet Usage Rates and Bicyclist Numbers Sustaining Head Injury*; August 1986. *Bicycle Helmet Usage Surveys of Primary and Secondary School Children, Adult Commuter and Recreational Cyclists*; May 1987.
29. DiGuiseppi CG, Rivara FP, Koepsell TD, Polissar L. Bicycle helmet use by children: evaluation of a community-wide campaign. *JAMA.* 1989;262:2256–2261.

Preventing and Postponing Death: Trends in Tennessee Infant Mortality

Joyce M. Piper, DrPH

ABSTRACT

Infants born in Tennessee between 1984 and 1988 experienced reduced mortality compared with those born in the 5 years previous. For all birthweight singleton infants, the reduction from 10.2 to 8.4 deaths per 1000 live births represents an 18% decline in mortality. The most impressive gain made was for very low birthweight (<1500 g) infants in the neonatal period, where mortality was reduced 24%. However, for these infants there was nearly a sixfold increase in the postneonatal mortality associated with prematurity-related causes.

(*Am J Public Health.* 1991;81:1046–1048)

Introduction

Evidence from the National Center for Health Statistics indicates that the rate of decline in infant mortality slowed during the 1980s.[1] Much of the progress made in the previous decade appears to be due to improvements in birthweight-specific mortality associated with care available at tertiary perinatal care facilities.[2–9]

One consequence of the impressive gains made by these facilities in preventing the deaths of very low birthweight (<1500 g) infants has been the postponement of some deaths from the neonatal to the postneonatal period.[10–13] These postponed deaths are thought to have contributed to the lack of decline in postneonatal mortality during the 1970s. Little is known about this effect in more recent data.

To determine whether reductions in Tennessee infant mortality have been achieved during the 1980s and to quantify any evidence of a postponement effect, we reviewed vital records data for 1979 through 1988.

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This paper was submitted to the journal November 26, 1990, and accepted with revisions April 2, 1991.

TABLE 1—Mortality Rates per 1000 Live Births By Birthweight Category for Tennessee Infants, 1979–1988

Birthweight Category	Mortality Rate Infants Born 1979–1983	 Infants Born 1984–1988	Difference in Rates (later vs earlier)	95% Confidence Interval
NBW(≥2500 g)				
Infant	5.0	4.0	−1.0	−1.3 to −0.7
Neonatal	2.1	1.6	−0.5	−1.8 to −0.3
Postneonatal	2.9	2.4	−0.5	−0.8 to −0.2
n	304 765	302 194	—	—
MLBW(1500–2499 g)				
Infant	31.1	26.9	−4.2	−7.6 to −0.8
Neonatal	19.8	16.4	−3.4	−6.1 to −0.7
Postneonatal	11.3	10.5	−0.8	−2.9 to 1.3
n	19 155	18 672	—	—
VLBW(<1500 g)				
Infant	372.7	296.8	−75.8	−98.4 to −53.4
Neonatal	332.4	251.9	−80.5	−102.2 to −58.8
Postneonatal	40.3	44.8	4.5	−5.1 to 14.1
n	3249	3481	—	—
Total				
Infant	10.2	8.4	−1.8	−2.3 to −1.3
Neonatal	6.4	5.1	−1.3	−1.7 to −0.9
Postneonatal	3.8	3.3	−0.5	−0.8 to −0.2
n	327 169	324 347	—	—

Note: NBW = normal birthweight; MLBW = moderately low birthweight; VLBW = very low birthweight.

Materials and Methods

Design, Population, and Data Sources

Two birth cohorts were studied: infants born in the period 1979 to 1983 and 1984 through 1988. Infants were grouped into two 5-year periods to dampen year-to-year rate fluctuations. Criteria for study entry included singleton live birth during 1979 through 1988, a birthweight of 500 to 5999 g, maternal race either White or Black, and Tennessee residence at birth and, for those infants who died, a death with recording of the vital events in Tennessee.

Infant death certificates were matched to corresponding birth certificates using a computerized linkage algorithm. Overall, birth information was available for 97.0% of the infants who died from the 1979 to 1983 birth cohort and for 96.7% of the infants who died from the later birth cohort. Investigation of a sample of the unlinked infant death certificates revealed that most were attributable to infant surname changes.

Study Definitions

Infants were classified into three birthweight categories: normal birthweight (NBW = ≥2500 g), moderately low birthweight (MLBW = 1500 to 2499 g), and very low birth weight (VLBW = <1500 g).

Analysis

The study analysis compared mortality rates for the two birth cohorts. Rate differences were calculated by subtracting rates of the 1979 to 1983 cohort from those of the 1984 to 1988 cohort. Confidence intervals (CI) of differences were calculated with standard techniques.[14]

We aggregated infant deaths into eight leading cause of death categories, as suggested by Dollfus et al.[15] This categorization scheme uses underlying cause, as reported on death certificates and coded with the International Classification of Disease, Ninth Revision (ICD-9), to group infant deaths that have similar etiologies or strategies for prevention.[15]

Results

In each of the three birthweight categories (NBW, MLBW, VLBW) there were reductions in the infant mortality rate for the later birth cohort, and these reductions were most pronounced for VLBW infants (Table 1). There were also reductions in neonatal mortality rates for all three groups, the greatest reduction occurring for the VLBW infants. However, reduction in postneonatal mortality was limited to NBW infants.

To examine these results further, we classified infant deaths for VLBW and MLBW infants using the categorization scheme previously described. For VLBW infants, there were reductions in the neonatal mortality rate for deaths associated with prematurity, birth asphyxia, and obstetric conditions (Table 2). However, for these infants, the postneonatal mortality rate for deaths associated with prematurity increased from 3.1 to 17.8 per 1000 live births. For MLBW infants, there were reductions in the neonatal mortality rate associated with prematurity (rate difference = −1.8, 95% CI of the difference −2.3–−1.3) and perinatal infections (rate difference = −2.1, 95% CI of the difference −3.3–−0.9). For these infants, there were no changes in the causes of postneonatal mortality.

Discussion

Our data show reduced infant mortality for infants born between 1984 and 1988 compared with those born in the five years previous. For infants of all birthweights the reduction from 10.2 to 8.4 deaths per 1000 live births represents an 18% decline in infant mortality. The most impressive gain made was for VLBW infants in the neonatal period, where the mortality rate declined from 332.4 to 251.9 deaths per 1000 live births, a 24% decrease. There was a small improvement in postneonatal mortality overall, but no improvement in this parameter for either MLBW or VLBW infants. For VLBW infants, there was nearly a sixfold increase in postneonatal mortality associated with prematurity-related deaths, suggesting that more of these fragile newborns are surviving the neonatal period only to die later of the complications of preterm birth.

It is unlikely that ascertainment bias could account for our findings, because we were able to link the same proportion of infant deaths to their birth certificates in the two time periods. The computerized scheme we used to group infant deaths of related causes was not subject to differential investigator surveillance. However, it is possible that changes in the attribution of underlying cause of death over time could have resulted in the increase of prematurity-related postneonatal deaths observed.

The postponement of the deaths of some VLBW infants from the neonatal to the postneonatal time period remains evident when data from the middle to late 1980s are compared with data from the early 1980s. These postponed deaths

TABLE 2—Neonatal and Postneonatal Mortality Rates by Cause of Death for Very Low Birthweight (VLBW) Infants Born in Tennessee, 1979 to 1988

Cause of Death	VLBW Infants Born in 1979–1983 Rate	(n)	VLBW Infants Born in 1984–1988 Rate	(n)	Difference in Rates (later vs earlier)	95% Confidence Interval
Neonatal						
Prematurity related	241.9	(786)	178.4	(621)	−63.5	−83.0 to −44.0
Perinatal infections	15.4	(50)	13.2	(46)	−2.2	−7.9 to 3.5
Birth asphyxia	15.7	(51)	9.2	(32)	−6.5	−11.9 to −1.2
Obstetric conditions	13.9	(45)	7.8	(27)	−6.1	−11.1 to −1.1
Congenital anomalies	20.9	(68)	19.2	(67)	−1.7	−8.4 to 5.0
SIDS[a]	—	(0)	—	(0)	—	—
Infections (nonperinatal)	2.8	(9)	2.9	(10)	0.1	−2.4 to 2.6
Injuries	0.3	(1)	0.3	(1)	0	−0.8 to 0.8
Other	21.5	(70)	21.0	(73)	−0.5	−7.4 to 6.4
Postneonatal						
Prematurity related	3.1	(10)	17.8	(62)	14.7	9.9 to 19.5
Perinatal infections	—	(0)	0.6	(2)	0.6	−0.2 to 1.4
Birth asphyxia	0.3	(1)	—	(0)	−0.3	−0.8 to 0.3
Obstetric conditions	—	(0)	—	(0)	—	—
Congenital anomalies	3.1	(10)	3.4	(12)	0.3	−2.4 to 3.0
SIDS[a]	6.2	(20)	4.9	(17)	−1.3	−4.8 to 2.2
Infections (nonperinatal)	9.2	(30)	5.7	(20)	−3.5	−7.6 to 0.6
Injuries	0.9	(3)	—	(0)	−0.9	−1.9 to 0.1
Other	17.5	(57)	12.3	(43)	−5.2	−17.6 to 7.2

[a] Sudden infant death syndrome.

probably represent an inevitable consequence of the application of technologically advanced medicine to multiple problems of high-risk newborns. Those concerned with improving infant health need to continue to examine both neonatal and postneonatal mortality, because early survival alone may not provide a complete picture. ☐

Acknowledgments

The author gratefully acknowledges the expert computer assistance of Mr. Edward F. Mitchel, Jr.

This study was supported in part by the Tennessee Department of Health and the Environment contracts Z-36073 and Z-36074.

References

1. Office of Maternal and Child Health. Progress toward achieving the 1990 objectives for pregnancy and infant health. *JAMA*. 1988;260:770–771.
2. Lee KS, Paneth N, Gartner LM, Pearlman MA, Gruss L. Neonatal mortality: an analysis of the recent improvement in the United States. *Am J Public Health.* 1980; 70:15–21.
3. Kleinman JC, Kovar MG, Feldman JJ, Young CA: A comparison of 1960 and 1973–74 early neonatal mortality in selected states. *Am J Epidemiol.* 1978;108:454–469.
4. Williams RL, Chen PN: Identifying the sources of the recent decline in perinatal mortality rates in California. *N Engl J Med.* 1982;306:207–214.
5. McCormick MC, Shapiro S, Starfield BH: The regionalization of perinatal services. *JAMA.* 1985;253:799–804.
6. Siegel E, Gillings D, Campbell S, Guild P. A controlled evaluation of rural regional perinatal care: impact on mortality and morbidity. *Am J Public Health.* 1985; 75:246–253.
7. Powell TG, Pharoah POD. Regional neonatal intensive care: bias and benefit. *Br Med J.* 1987;295:690–692.
8. Paneth N, Kiely JL, Wallenstein S, Marcus M, Pakter J, Susser M. Newborn intensive care and neonatal mortality in low birthweight infants. *N Engl J Med.* 1982; 307:149–155.
9. Gortmaker S, Sobel A, Clark C, Walker DK, Geronimus AB. The survival of very low birth weight infants by level of hospital of birth: a population study of perinatal systems in four states. *Am J Obstet Gynecol.* 1985;152:517–524.
10. Hack M, Merkatz IR, Jones PK, Fanaroff AA. Changing trends of neonatal and postneonatal deaths in very-low birthweight infants. *Am J Obstet Gynecol.* 1980;137:797–800.
11. Zdeb MS. Differences in trends of postneonatal mortality by birthweight in upstate New York, 1968–79. *Am J Public Health.* 1982;72:734–736.
12. Buehler JW, Hogue CJR, Zaro SM. Postponing or preventing deaths? Trends in infant survival, Georgia, 1974 through 1981. *JAMA.* 1985;253:3564–3567.
13. Friede A, Rhodes PH, Guyer B, Binkin NJ, Hannan MT, Hogue CJR. The postponement of neonatal deaths into the post neonatal period: evidence from Massachusetts. *Am J Epidemiol.* 1988;127:161–170.
14. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research: Principles and Quantitative Methods.* Belmont, Calif: Lifetime Learning Publications;1982:297.
15. Dollfus C, Patetta M, Siegel E, Cross AW. Infant mortality: a practical approach to the analysis of the leading causes of death and risk factors. *Pediatrics.* 1990;86:176–183.

A Comparison of Breast-Feeding Data from the National Surveys of Family Growth and the Ross Laboratories Mothers Surveys

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ABSTRACT

This study compares rates of breast feeding reported in the National Surveys of Family Growth with those from the Ross Laboratories Mothers Surveys. Both surveys have documented rates of breast feeding over the last 30 or more years. Despite differences in survey methodology, both surveys document similar long-term trends in breast feeding. The similarities of rates in breast feeding also are evident across several maternal sociodemographic characteristics. We conclude that both surveys produce reliable and useful estimates of breast feeding. (*Am J Public Health.* 1991; 81:1049–1052)

Introduction

The principal sources of information on breast-feeding trends in the United States are the National Surveys of Family Growth (NSFG) and the Ross Laboratories Mothers Surveys (RLMS). These two surveys have documented trends in breast feeding over the last three decades. This study compares reported rates of breast feeding in the NSFG with those in the RLMS between 1955 and 1987 and compares the rates in 1987 according to selected sociodemographic characteristics.

We found that despite differences in survey methodologies and design, both surveys document similar trends in breast-feeding, and these similarities are evident across sociodemographic characteristics. The similarities of breast-feeding rates bear witness to the usefulness of the surveys in documenting the incidences and long-term trends of breast-feeding practices in the United States.

Methods

National Surveys of Family Growth

Information on breast-feeding has been collected by the National Center for Health Statistics in four NSFGs: Cycle I (1973), Cycle II (1976), Cycle III (1982), and Cycle IV (1988).[1–5] The surveys were designed to provide information on factors affecting childbearing, such as contraception, sterilization, infertility, and related aspects of maternal and infant health.[6] In addition to a series of detailed demographic questions, during a face-to-face personal interview, each mother was asked, "When your child was an infant, did you breast-feed (him/her) at all?" (i.e., ever-breast-fed).

Because the surveys focused on fertility, the NSFG included samples that were representative of the national population of civilian noninstitutionalized women in their reproductive years (ages 15 through 44 years). Cycles I and II had sample sizes of 9800 and 8600 women, respectively, who were currently or previously married. Cycle III included 7969 women of all marital statuses and women living in group quarters. The Cycle IV target population was expanded to include women living in Alaska and Hawaii. In Cycle IV, 8450 women were selected; this sample was drawn from households interviewed between October 1985 and March 1987 for another NCHS survey, the National Health Interview Survey (NHIS). The response rate to the NHIS was 96%, and the response rate to Cycle IV of the NSFG was 82% of the women subsampled from the NHIS. Thus, the total NSFG Cycle IV response rate is approximately 79%.

Ross Laboratories Mothers Surveys

Ongoing surveys by Ross Laboratories have documented trends in breast feeding in the United States since 1955.[7–12] The RLMS is a large, national mail survey designed to determine patterns of infant feeding during the first 6 months of life.

Each quarter, questionnaires are mailed to mothers when their infants reach 6 months of age. Mothers are asked to recall the type of milk fed to their infant immediately after birth, typically in the hospital, and during each month of the first 6 months of life.

The sample is derived from a list of births that consists of approximately 70% to 82% of all new mothers in the United States. The list includes names from hospital sources, county records of birth reg-

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This paper was submitted to the journal November 8, 1990, and accepted with revisions April 25, 1991.

TABLE 1—Incidence of Breast Feeding, 1955–1987. National Surveys of Family Growth (NSFG) Compared with Ross Laboratories Mothers Survey (RLMS)

	NSFG	RLMS	
Year	Breast Fed at Any Time	In Hospital	1 Week
1955–1959	32.0	—	28.7
1960–1964	27.0	—	27.4
1965–1969	25.0	28.1	26.0
1970	23.0	26.5	24.9
1971	25.0	24.7	22.8
1972	22.0	28.1	25.9
1973	25.0	28.9	26.8
1974	30.0	32.2	29.6
1975	34.0	35.5	33.4
1976–1977	42.2	43.2	40.9
1978–1979	44.3	48.8	47.4
1980–1981	52.5	56.4	56.2
1981	57.9	57.8	56.4
1982	59.1	61.9	60.5
1983	55.3	58.4	57.0
1984[a]	54.2	59.2	57.6
1985[a]	55.6	57.3	55.6
1986[a]	53.5	56.3	54.9
1987[a]	56.3	56.1	54.7

[a] RLMS breast-feeding data have been adjusted to reflect time that survey was mailed (adjusted year = last half of year + first half of following year; see text).

istrations, photography and entrepreneurial services, and newspapers.

Throughout the past few years, the number of questionnaires mailed to new mothers has increased substantially. In 1984, 56 894 questionnaires were mailed and 30 694 (54%), or 0.8% of annual births, were returned. In 1987, 154 958 questionnaires were mailed and 83 240 (54%), or 4.1% of annual births, were returned.

The collected infant-feeding data are then weighted by subclass means in the sample to account for differing responses and coverage rates. The subclasses were defined by the variables that have historically been associated with breast-feeding: maternal region of residence, ethnic background, age, and education. The weights were assigned using continually updated information from the US Bureau of the Census and National Center for Health Statistics.[13–16]

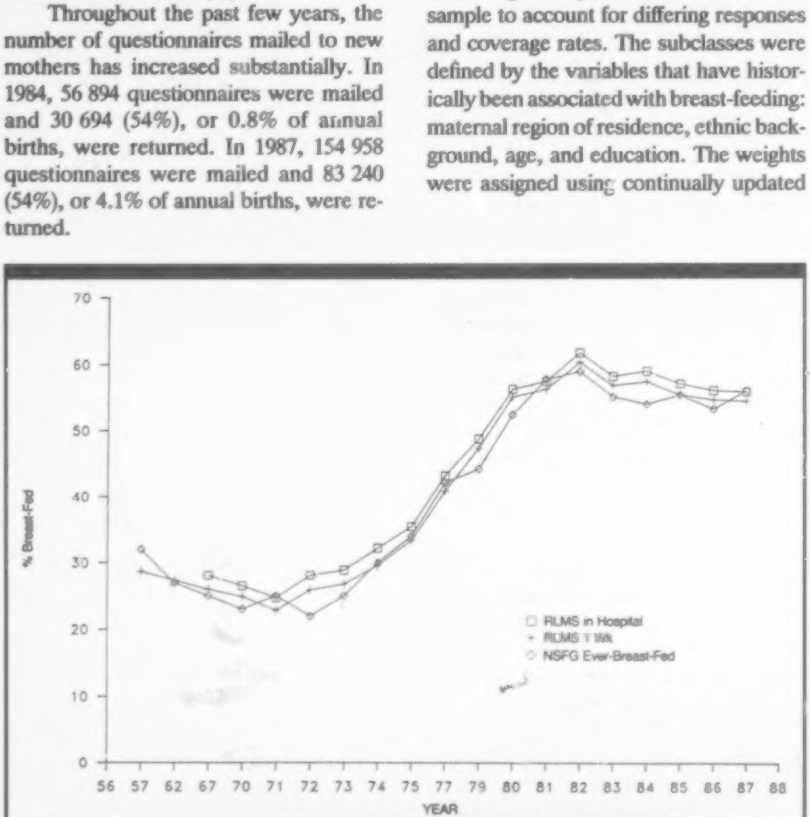

FIGURE 1—Incidence of breast feeding, 1957–1987: National Surveys of Family Growth (NSFG) compared with Ross Laboratories Mothers Survey (RLMS). The term "breast feeding" is defined as the exclusive use of human milk or the use of human milk with a supplemental bottle (i.e., formula or cow's milk).

Data Analyses

Breast-feeding rates reported at two time periods in the RLMS (in the hospital and at 1 week of age) were compared with the ever-breast-fed rates reported in the NSFG. We also considered rates of breast feeding in 1987 (most recent data in both surveys) according to the following sociodemographic characteristics: maternal age, parity, ethnic background, education, region of residence, and birthweight.

Because the RLMS questionnaire is mailed to mothers when their infants are 6 months of age, to produce yearly estimates that are comparable to those provided in the NSFG, it was necessary to adjust for the time the RLMS survey was mailed. For example, the adjusted data for 1987 represent information collected during the last half of 1987 and the first half of 1988 (Table 1). These adjusted data may differ slightly from previously published data that have not been adjusted.[11]

Since the data in the RLMS and NSFG were obtained through complex sample designs, it was necessary to use specific statistical techniques that incorporated the sample weights for the computation of estimated breast-feeding percentages and standard errors.[6,17,18] Differences between rates of breast-feeding in the two surveys were tested for statistical significance using a z test ($P<.05$).

Results: Trends in Breast Feeding

Both surveys show a small decline in the incidence of breast feeding from 1955 to the early 1970s, followed by an increase until 1982, when the incidence of breast feeding reached its peak (Figure 1). Between 1957 and 1988, the two surveys demonstrate an approximate twofold net increase in the incidence of breast feeding (32.0% to 56.3% in NSFG; 28.7% to 54.7% in RLMS at 1 week of age). Since 1982, as reported in RLMS, the incidence in the initiation of breast feeding and breast feeding at 1 week of age steadily declined (61.9% to 56.1% in the hospital; 60.5% to 54.7% at 1 week of age). The NSFG ever-breast-fed data showed a statistically nonsignificant net decline between 1982 and 1987 (59.1% to 56.3%).

Since 1955, at each year, differences between the two surveys in rates of breast-feeding were not large. Over time, the av-

TABLE 2—Incidence of Breast-Feeding: Ross Laboratories Mothers Survey (RLMS) Compared with 1988 National Survey of Family Growth (NSFG) for 1987

Characteristics	RLMS[a] N	RLMS[a] Breast Feeding (%)	NSFG[b] N	NSFG[b] Breast Feeding (%)	Difference	Standard Error of Difference	95% Confidence Interval of Difference
Total United States	83 240	54.7	563	56.3	−1.6	3.2	−7.9, 4.7
Ethnicity[d]							
Black (non-Hispanic)	10 151	25.3	195	23.1	2.2	4.0	−5.6, 10.0
White (non-Hispanic)	66 136	61.1	338	61.0	0.1	3.5	−6.8, 7.0
Hispanic	5 423	49.7	50	41.8	7.9	8.1	−8.0, 23.8
Maternal education[d]							
High school or less	36 043	43.9	334	44.6	−0.7	4.6	−9.7, 8.3
Some college	46 816	72.0	229	71.6	0.4	3.5	−6.5, 7.3
Maternal age (years)[d]							
<20	4 544	30.5	59	26.1	4.4	6.6	−8.5, 17.3
20–24	19 389	47.1	168	50.2	−3.1	7.3	−17.4, 11.2
25–29	31 425	60.9	185	60.1	0.8	4.1	−7.2, 8.8
30–34	20 677	66.4	104	61.5	4.9	5.6	−6.1, 15.9
>34	7 068	66.7	47	78.1	−11.4	6.9	−24.9, 2.1
Region							
Northeast	14 976	51.6	110	68.3	−16.7	5.9	−28.3, −5.1[c]
South	27 761	46.0	201	41.1	4.9	5.2	−5.3, 15.1
Midwest	21 720	52.6	137	48.6	4.0	4.4	−4.6, 12.6
West	18 783	72.9	115	74.1	−1.2	5.3	−11.6, 9.2
Residence							
Urban	62 975	55.3	449	57.4	−2.1	3.7	−9.4, 5.2
Rural	20 265	52.9	114	52.9	0.0	5.2	−10.2, 10.2
Parity[d]							
Primiparous	41 098	55.3	188	56.3	−1.0	4.7	−10.2, 8.2
Multiparous	41 028	54.2	375	56.3	−2.1	4.3	−10.5, 6.3
Birthweight (g)[d]							
<2500	4 113	38.4	43	32.7	5.7	10.1	−14.1, 25.5
>2500	75 649	56.1	518	57.7	−1.6	3.3	−8.1, 4.9

[a] Breast feeding reported at 1 week of age.
[b] Infants who have been breast fed at any time.
[c] $P<.05$.
[d] The sample sizes for these demographic subsets do not add to total because of unit nonresponse.

erage difference between the NSFG ever-breast-fed and the RLMS in-hospital breast-feeding rates was 2.6%; differences ranged from 0.3% in 1971 to −6.1% in 1972. At each year, the RLMS in-hospital breast-feeding rates tended to be higher than the NSFG ever-breast-fed rates.

Differences between the NSFG ever-breast-fed and the RLMS breast feeding at 1 week of age rates were smaller than differences between the NSFG ever-breast-fed and RLMS in-hospital rates; the average difference was only 0.6%, and differences ranged from 3.3% in the period 1955 to 1959 to −3.9% in 1972 (Figure 1).

As shown in Table 1, both surveys demonstrated that in 1987, breast-feeding was relatively high among women who were White, older in age, college educated, living in the urban and western regions of the United States, and who had an infant of normal birthweight. Also, according to both surveys, the lowest rates of breast feeding were among women who were Black, with less than a college education, residing in the rural and southern regions of the country, younger in age, and who had an infant of low birthweight.

The *z*-test analyses indicated that across the socioeconomic subgroups considered, except for the northeast region, differences in breast-feeding rates between the two surveys were not statistically significant (Table 2).

Discussion

The present study indicates that over the last 30 or more years, differences in breast-feeding rates between the NSFG and RLMS surveys were small. Both surveys agree in showing a decline in breast-feeding from 1955 to the early 1970s, followed by an upward trend until 1982. They also agree in demonstrating that, across several selected maternal characteristics, certain subgroups of mothers are more likely to breast-feed than others.

The NSFG ever-breast-fed rates and the RLMS breast feeding at 1 week of age rates were comparable (Table 1). The RLMS in-hospital breast-feeding rates averaged approximately 2% to 3% higher than the NSFG ever-breast-fed rates. In the NSFG, mothers were asked if they ever breast-fed their infant, whereas in the RLMS, mothers were asked to indicate the kinds of milk they fed to their infant. A possible explanation for the lower in-hospital breast-feeding rates in the NSFG is that respondents who fed breast milk to their infant in the hospital but not after leaving may have considered such a brief encounter a nonexperience. In contrast, mothers in RLMS were not asked if they actually "breast-fed"; they may have given their infants a bottle containing expressed human milk. Because the questions used to define breast-feeding were not strictly comparable, the observed differences in the in-hospital breast-feeding rates may reflect variation in the manner in which mothers define breast-feeding.

Except for breast-feeding in the northeast region of the United States, across all other sociodemographic subgroups of mothers, differences in breastfeeding rates between the two surveys were not statistically significant. Considering the many comparisons that were made between these two surveys on mothers' characteristics, the finding of a single, statistically significant difference is not surprising and probably warrants little attention.

The principal sources of data on long-term trends in breast-feeding are the National Surveys of Family Growth and the Ross Laboratories Mothers Survey. The similarities in estimates support the usefulness of these surveys for documenting rates of breast-feeding in the United States. ☐

Acknowledgments

We gratefully acknowledge the assistance of Gilbert A. Martinez, who provided valuable insight into trends in infant-feeding practices. We also thank Steven Botman of NCHS for statistical advice.

References

1. Hendershot GE. *Trends in Breast Feeding.* Hyattsville, Md: National Center for Health Statistics; 1980. US Dept of Health and Human Services publication 8-1250.
2. Hirschman C, Butler M. Trends and differentials in breast feeding: an update. *Demography.* 1981;18:39–54.
3. Hirschman C, Hendershot GE. *Trends in Breastfeeding among American Mothers.* Hyattsville, Md: National Center for Health Statistics; 1979. US Dept of Health, Education, and Welfare publication (PHS) 79-1979. (Vital and Health Statistics, Series 23, No. 3).
4. Hirschman C, Sweet JA. Social background and breastfeeding American mothers. *Soc Biol.* 1974;21:39–57.
5. Hendershot GE. Trends in breast-feeding. *Pediatrics.* 1984;74(suppl):591–602.
6. National Center for Health Statistics. *National Survey of Family Growth, Cycle IV: Sample Design, Weighting, Imputation, and Variance Estimation.* Hyattsville, Md; September 1989. Data Evaluation and Methods Research Series 2.
7. Martinez GA, Nalezienski JP. The recent trend in breast-feeding. *Pediatrics.* 1979;64:686–692.
8. Martinez GA, Nalezienski JP. 1980 update: the recent trend in breast-feeding. *Pediatrics.* 1981;67:260–263.
9. Martinez GA, Dodd DA, Samartgedes JA. Milk feeding patterns in the United States during the first 12 months of life. *Pediatrics.* 1981;68:863–868.
10. Martinez GA, Dodd DA. 1981 milk feeding patterns in the United States during the first 12 months of life. *Pediatrics.* 1983;71:166–170.
11. Martinez GA, Krieger FW: 1984 milk-feeding patterns in the United States. *Pediatrics.* 1985;76:1004–1008.
12. Ryan AS, Martinez GA. Breast-feeding and the working mother: a profile. *Pediatrics.* 1989;83:524–531.
13. National Center for Health Statistics. *Advance Report of the Final Natality Statistics*, 1982. Hyattsville, Md: Public Health Service; September 1984. Monthly Vital Statistics Report, Vol. 33, No. 4(suppl).
14. National Center for Health Statistics. *Advance Report of the Final Natality Statistics, 1987.* Hyattsville, Md: Public Health Service; June 1989. Monthly Vital Statistics Report, Vol. 38, No. 3(suppl).
15. US Bureau of the Census. *Fertility of American Women: June 1983 (Advance Report).* Washington, DC: US Government Printing Office; April 1984. Population Characteristics, Series P-20, No. 386.
16. US Bureau of the Census. *Fertility of American Women: June 1986* (Advance Report). Washington, DC: US Government Printing Office; June 1987. Population Characteristics, Series P-20, No. 427.
17. Statistical Analysis System Institute. *SAS User's Guide: Statistics*. 5th ed. Cary, NC; 1985.
18. Efron B. Bootstrap methods: another look at the jackknife. *Ann Statist.* 1979;7:1–26.

Risk Factors for Childhood Homicides in Ohio: A Birth Certificate–Based Case-Control Study

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ABSTRACT

Death certificates of children less than 8 years of age who were killed between 1979 and 1986 were linked to their Ohio birth certificates and compared with those of Ohio children born in 1983 (controls). Having an unmarried mother increased risk of homicide almost fivefold (odds ratio 4.87). Having a teenage mother, a mother who had not graduated from high-school, and being of Black race or low birthweight each increased the risk by approximately threefold. Increases in the proportion of children born to unmarried mothers may contribute to increases in childhood homicide rates. (*Am J Public Health*. 1991;81:1052–1054)

Introduction

Rates of death by homicide in children have increased markedly in the United States and in Ohio in the last 30 years, particularly for children under age 5.[1–3] While many of the risks associated with childhood homicide are known,[4–11] we undertook a case-control study of homicides in Ohio children under the age of 8, to identify risk factors and to clarify the associations among those factors.

Methods

Cases were taken from death certificates of child homicide cases (International Classification of Disease, revision 9 [ICD-9] codes E-960 through E-969) from the years 1979 through 1986, of Ohio residents who were born during those same years. For each case, the birth certificate was sought in the Ohio birth certificate file, matching on last name, first three letters of first name, and date of birth. Con-

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This paper was submitted to the journal November 6, 1990, and accepted with revisions February 20, 1991.

TABLE 1—Characteristics of Ohio Child Homicide Cases, Ages 0 to 7 Years, 1979–1986, and 1983 Ohio Birth Cohort Controls

Characteristic	Case	Control
Born out of wedlock (%)	54.2	19.6
Black race (%)	38.0	15.1
Low birthweight (%)	16.2	6.7
Mother not a high school graduate (%)	47.4	20.8
Pregnancy complications (%)	12.0	9.6
Illness in pregnancy (%)	4.2	3.4
Singleton birth (%)	96.5	97.9
Male (%)	60.6	51.2
Mother's age (mean years)	21.9	25.3

TABLE 2—Odds Ratios (ORs) and Confidence Intervals (CI) for Selected Risk Factors for Child Homicide in Ohio Children, Ages 0 to 7 Years, 1979–1986, Compared with 1983 Ohio Births as Controls

Risk Factor	OR[a]	95% CI[b]
Born out of wedlock	4.87[a]	3.45–6.89
Black race	3.52[a]	2.47–5.02
Low birthweight	2.75[a]	1.71–4.38
Mother not a high-school graduate	3.45[a]	2.43–4.88
Pregnancy complications	1.32	0.77–2.24
Illness in pregnancy	1.29	0.51–3.02[b]
Singleton birth	0.57	0.22–1.57[b]
Labor complications	1.15	0.78–1.68
Metro county resident	1.52[a]	1.04–2.21
Congenital anomalies	0.55	0.03–3.64[b]
Teenaged mother	3.20[a]	2.23–4.60

[a] Statistically significant

[b] Less than five frequencies per cell

trols, also obtained from Ohio birth certificates, were all children born live to Ohio residents in 1983.

Factors examined in this study included marital status of the victim's mother at the time of the victim's birth, race (Black vs White), educational attainment of the mother (<12 years vs ≥12 years), county type (metropolitan vs nonmetropolitan), birthweight (<2500 g vs ≥2500 g), mother's age (teenager or ≥20), presence or absence of birth complications, illness during pregnancy, labor complications, multiple birth, and congenital anomalies.

Odds ratios (ORs) were calculated for each of these potential risk factors. Those risk factors associated with the outcome in a univariate analysis were examined in a stratified analysis, two at a time. Cornfield's method was used to calculate 95% confidence limits around the odds ratios.[12]

Results

There were 189 children who were born and died during the study period; birth certificates were identified for 140, or almost 75%. Of these, 134, or 96%, were under age 5. Mean age at death was 1.1 years. Children for whom birth certificates could not be found were more likely to be Black (48% vs 39% for whom certificates were found), metropolitan residents (78% vs 30%), and/or less than 28 days old at the time of death (29% vs 7%).

Cases were more likely to be Black, male, and of low birthweight than controls (Table 1). Likewise, case mothers were younger at the time of the child's birth and less likely to be married or to have a high school diploma.

Univariate ORs (Table 2) were highest for being born out of wedlock, Black race, having a mother who did not finish

TABLE 3—Crude and Stratified Odds Ratios (ORs) for Homicide, Ohio Cases under 7 Years of Age from 1979 to 1986, Compared with All 1983 Ohio Births

Risk Factor	Out-of-Wedlock Birth	Black	Teenaged Mother	Metro County Residence	High School Nongraduate	Low Birthweight
Crude	4.87[a]	3.52[a]	3.20[a]	1.52[a]	3.45[a]	2.75[a]
Stratified by:						
Legitimacy						
Legitimate	—	1.51	3.24[a]	0.97	2.41[a]	2.77[a]
Out of wedlock	—	1.84[a]	1.27	1.47	1.82	1.77
Race						
Black	4.29[a]	—	1.45	2.44	2.88[a]	2.72[a]
White	3.84[a]	—	4.06[a]	0.98	3.14[a]	1.70
Mother's age						
<20 years	2.04[a]	1.48	—	1.28	1.68	0.85
≥20 years	5.19[a]	4.13[a]	—	1.65[a]	3.16[a]	3.95[a]
County type						
Metro	5.37[a]	3.54[a]	2.96[a]	—	4.65[a]	2.77[a]
Nonmetro	3.54[a]	1.42[a]	3.82[a]	—	1.78	2.16
Mother's education						
Not a high school graduate	4.41[a]	2.73[a]	1.58	2.68[a]	—	1.88
High school graduate	3.33[a]	2.96[a]	2.98[a]	1.03	—	2.70
Birthweight						
Low	3.10[a]	4.72[a]	0.84	1.72	2.49	—
Normal	4.86[a]	2.98[a]	3.89[a]	1.34	3.58[a]	—

[a] Lower bound of 95% confidence limit does not include 1.0.

TABLE 4—Homicide Deaths and Out-of-Wedlock Births of Ohio Children by Race and Specific Years

	No. of Homicide Deaths under Age 5 Years	Homicide Rate per 100 000 Deaths under Age 5 Years	All Ohioans born to unwed Mothers (%)
White			
1970	17	2.1	5.8
1980	19	2.8	11.0
1986	12	1.7	15.7
Black			
1970	7	7.2	38.3
1980	9	9.3	56.8
1986	20	19.9	64.0

high school, having a teenaged mother, having a birthweight less than 2500 g, and living in a metropolitan county.

The effect of having an unmarried mother on risk of homicide was strong in all subgroups examined (Table 3), but was greatest among children whose mothers were age 20 or older, and/or lived in a metropolitan area, and among children who were of normal birthweight.

The crude risk of homicide associated with Black race was 3.52 (Table 3), but the risk fell to between 1.5 and 1.8 when separate strata of children born to married and unmarried mothers were examined. Thus, the apparent effect of Black race on risk of homicide is largely explained by the fact that a larger proportion of Black infants were born to unmarried mothers. The effect of Black race on risk of homicide is greatest in children residing in metro counties, in low-birthweight children, and in children with mothers over age 20, and varies little by educational status of the mother.

The crude risk of dying from homicide is increased approximately 3.2-fold when the child's mother is a teenager at the time of the child's birth. The magnitude of this increase in risk is greatest when the child is otherwise at lower risk.

The crude OR for metropolitan residence is only of modest magnitude (1.52), but the increase in risk is much greater when the mother is not a high school graduate or when the mother is Black.

Children born to mothers who did not finish high school (a proxy for low socioeconomic status[13–15]) are approximately 3.45 times more likely to become homicide victims than are children born to high school graduates. This OR estimate appears to be confounded in part by effects of race, mother's age, and marital status, as the stratum-specific estimates for both strata of all three variables were noticeably less than the crude OR.

Low birthweight was a significant risk factor for homicide in most subgroups. Low birthweight is more strongly associated with risk of homicide among Black than White infants, and among children whose mothers were married and age 20 or older at the time of their child's birth.

Discussion

Many of the expected relationships found in other studies were observed in these data: children of younger, poorly educated mothers, Black children, children of low birthweight, children whose mothers were unmarried at the time of the child's birth, and children who resided in metropolitan areas were all more likely to be killed than other children. In general, these variables appear to be independently associated with the risk of homicide. Variables associated with the pregnancy were not good predictors for risk of homicide, nor were congenital malformations. The effect of race is largely explained by the high prevalence of Black infants with unmarried mothers. Similarly, the crude effect of low educational attainment of the mother on risk of homicide appears to be explained in part by the fact that these mothers are more likely to be unmarried. A weaker effect of having an unmarried or teenaged mother on risk of homicide was seen when the infant was otherwise at lower risk. This suggests that other social supports in the child's environment may reduce the risk of homicide.

The consistent association of homicide risk with being born to an unmarried mother is striking in these data. It is unclear whether this finding represents a causal relationship, or whether both increased risk of homicide and being born to an unmarried mother are consequences of some other risk factor that was not captured in our study. If there is a causal relationship, we can expect the incidence of childhood homicide to continue to increase, because the percentage of Ohio children born to unmarried mothers has increased dramatically in recent years (Table 4).

Children have many health and social problems, which makes the causes of childhood homicide complex. However, interventions to help teenagers finish high school, begin careers, and find permanent life partners before becoming parents would probably help prevent childhood homicide and abuse.[16] Such interventions are, in any case, desirable for other reasons. The strong associations between having a young or unmarried mother and the risk of homicide suggest that interventions to prevent homicides in children should focus on these social variables. □

References

1. Jason J. Child homicide spectrum. *Am J Dis Child.* 1983;137:578–581.
2. Ordog G, Wasserberger J, Schatz I, et al. Gunshot wounds in children under 10 years of age: a new epidemic. *Am J Dis Child.* 1988;142:618–622.
3. Ohio Department of Health; Bureau of Vital Statistics, Data Analysis by Special Studies Branch, Bureau of Epidemiology. Columbus Ohio; no year.
4. Simons B, Downs EF, Hurster MM, Archer M. Child abuse: epidemiologic study of medically reported cases. *NY State J Med.* November 1966;2783–2788.
5. Kaplun D, Reich R. The murdered child and his killers. *Am J Psychiatry.* 1976;133:809–813.
6. Jason J, Gilliland JC, Tyler CW. Homicide as a cause of pediatric mortality in the United States. *Pediatrics.* 1983;72:191–197.
7. Rosenberg M, Gelles R, Holinger P, et al. Violence: homicide, assault, and suicide. *In:* Amler R, Dull H., eds. *Closing the Gap: The Burden of Unnecessary Illness.* New York: Oxford University Press; 1987.
8. Baldwin J, Oliver J. Epidemiology and family characteristics of severely abused children. *Br J Prev Soc Med.* 1975;29:205–221.
9. Hollander N. Physical abuse as a predictor of child homicide. *Tex Med.* 1986;82:21–23.
10. Scott PD. Parents who kill their children. *Med Sci Law.* 1973;13:120–126.
11. Muscat J. Characteristics of childhood homicide in Ohio, 1974–1984. *Am J Public Health.* 1988;78:822–824.
12. Kleinbaum D, Kupper L, Morganstern H. *Epidemiologic Research.* New York: Van Nostrand Reinhold Co; 1982:304–305.
13. Winkleby M, Fortmann S, Barrett D. Social class disparities in risk factors for disease: eight-year prevalence patterns by level of education. *Prev Med.* 1990;19:1–12.
14. Mueller C, Parcel T. Measures of socioeconomic status: alternatives and recommendations. *Child Dev.* 1981;52:13–30.
15. Kleinman J, Kessel S. Racial differences in low birthweight: trends and risk factors. *N Engl J Med.* 1987;317:749–753.
16. Schorr, L. *Within Our Reach.* New York: Anchor Press; 1988.

Costs and Benefits of Implementing Child Survival Services at a Private Mining Company in Peru

ABSTRACT

Costs and savings of child health services were studied in a private mining company in Peru. Despite considerable outlays for medical services, few children under age 5 were vaccinated, and half of their illnesses went untreated. Children who were attended at the company clinic usually received unnecessary medication. As a result of the study, the company hired additional staff to provide integrated maternal-child preventive health care and family planning and contracted for intensive training and periodic on-site supervision. In less than 2 years, vaccination coverage reached 75%, and virtually all children under age 1 were enrolled in growth monitoring. Prescriptions were reduced by 24%, including a 67% drop in antimicrobials. The cost of the new services was $13 200 for the first 2 years. Approximately $6800 has been saved in pharmaceuticals prescribed for respiratory infection and diarrhea. Recently, two more mines adopted maternal and child health and family planning services. It is hoped that cost-benefit arguments will encourage other companies to incorporate aggressive child survival measures into their health plans. (*Am J Public Health.* 1991;81;1055–1057)

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Introduction

As public resources stagnate, developing countries must find alternative sources of financing for primary health. One underutilized resource is the commercial sector. Approximately 10% of the 1984 health expenditures in Peru were covered by payments made by employers,[1] a source that could be tapped for preventive health care.

Most commercial health plans only cover curative care. The present study examined whether expenditures for curative child health care could be reduced through preventive interventions and better prescription practices, while maintaining or improving quality of care. The study site was a privately owned Peruvian mine that provides medical coverage to approximately 800 workers and 4000 dependents, including 900 children under age 5.

Despite company investments of $1.7 million in welfare and medical care, the population showed high morbidity. A prospective cost-benefit analysis found that family planning and child survival services could improve health status, at a cost that would be compensated by savings in other services averted. The company implemented the recommended services; this report summarizes the findings of the child survival component.

Methods

Baseline conditions were assessed during 1986 and 1987. Data included company records and household and clinic surveys. The household survey was conducted in July 1986, with a random sample of 220 miners' wives. The clinic survey was conducted in March 1987 and included 132 consultations with children under age 5. Preventive health services were added in 1988. A follow-up conducted in late 1989 included service statistics, a campwide census, and a clinical survey that captured 206 visits by children under age 5. Wholesale costs of medications prescribed were calculated at baseline and at follow-up.

Results

Baseline Conditions

Infant and child health were precarious at the start of this study. Infant mortality was estimated at 120 deaths per 1000 live births. Contributing factors included lack of well-baby care, low vaccination coverage, high incidence of diarrhea and respiratory infection, and overmedication and use of inappropriate medications.

Lack of well-baby care. All consultations surveyed were motivated by illness. Physicians reported that they had no time for well-baby care and that mothers would not use it if it were offered.

Vaccination. The household survey found that fewer than 5% of the children ages 1 to 4 were completely vaccinated. No child was vaccinated during an observed clinic visit. A quarter of the children over age 1 who had not been vaccinated spontaneously reported having had measles.

Morbidity. Of the children surveyed, 44% reported diarrhea and/or cough in the last 3 days; 24% reported diarrhea and 29% cough. Half the reported cases of diarrhea and/or cough did not seek treatment. Nevertheless, children under age 5, although they make up only 17% of the population, accounted for 50% of all clinic visits. Etiology was not determined, but the high incidence of blood and/or mucus in the stool accompanied by vomiting suggests shigellosis.[2] Respiratory infection accompanied by diarrhea is characteristic of rotovirus.

Overmedication. International norms suggest minimal medication for di-

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This paper was submitted to the journal July 11, 1990, and accepted with revisions March 6, 1991.

TABLE 1—Mean Number of Medications Prescribed by Physician's Diagnosis for Children under Age 5, 1987 vs 1989

	Diagnosis							
	ARI[a] Alone		Diarrhea Alone		ARI and Diarrhea		Others	
Medication	1987	1989	1987	1989	1987	1989	1987	1989
Antimicrobials	1.3	0.6	1.8	0.4	1.4	0.3	0.7	0.5
Symptomatics	1.8	2.0	1.2	1.4	2.1	2.0	1.0	0.8
Others	0.7	0.2	0.5	0.6	0.5	0.4	1.5	0.8
Total medications	3.7	2.9	3.5	2.4	4.0	2.7	3.2	2.0
No. of cases	47	85	24	44	28	14	21	21

[a]ARI = acute respiratory infection.

TABLE 2—Projected and Actual Costs and Estimated Cost Savings of Child Survival Activities

		Actual Costs		Estimated Savings	
Item	Projected Costs	Year 1	Year 2	Year 1	Year 2
Training and supervision	1500	1657	1946		
Salary and benefits	6300	2807	6666		
Equipment and materials	265	154	0		
Total	8065	4618	8612	1618	5147

arrhea and respiratory infection: oral rehydration and ampicillin for diarrhea with blood or mucus, acetaminophen and benzathine penicillin for moderate respiratory infection, etc. Nevertheless, every patient under age 5 received at least 2 and on average 3.7 medications, regardless of symptoms. Prescriptions for respiratory infection and diarrhea exceeded international recommendations (see Table 1). Prescriptions were classified according to the *Physician's Desk Reference*[3] and its Peruvian counterpart, *Diccionario de Especialidades Farmaceuticas*.[4]

Intervention

The study recommended that the mine hire an additional physician and nurse to provide well-baby and maternal care, and follow World Health Organization norms[5] for treating acute respiratory infection (ARI) and diarrhea. First-year costs were estimated at $8065; projected savings in pharmaceuticals ranged from $10 600 to $12 600 per year.

The company hired extra staff and contracted for training and on-site supervision. Services began in May 1988, in coordination with a national vaccination campaign. During the first quarter of 1989, standardized diagnostic and treatment protocols for ARI and diarrhea were adopted, and management contracted for a second year of supervision.

Program Impact

Vaccination coverage. The census found 75% coverage among children ages 1 to 4. Virtually all children under age 1 had been appropriately vaccinated.

Well-baby care. Of all clinic visits made in November 1989, 16% were for well-baby care, compared with none at baseline. Approximately 90% of children under age 1 and 35% of children ages 1 to 4 had been enrolled in growth monitoring. Thirty-nine children with second- and third-degree malnutrition were enrolled in nutritional rehabilitation; 31% returned to normal.

Treatment of diarrhea and respiratory infection. During 1989 the clinic treated 2258 cases of respiratory infecion and 1233 cases of diarrhea among children under age 5. As shown in Table 1, physicians prescribed on average one fewer medication per consultation; mean number of antimicrobials prescribed for ARI and diarrhea decreased from 1.5 to 05.

Costs and Savings

During the first 2 years, the company invested $13 200 in new child services at a cost per capita of less than $7.50 per year, compared with baseline per capita expenditures of $160 for curative care. After 18 months, annual prescription savings were approximately $5000 (January 1990 prices). Assuming linear trends, savings during the first 2 years totaled $6800 without considering improved health status due to better vaccination and nutrition practices. Table 2 presents program costs and estimated savings.

Discussion

Most child survival efforts focus on children outside the formal health system. However, many children inside the system also do not have access to preventive care. Furthermore, access to curative services does not guarantee use of services or that appropriate treatment will be given if sought. Consequently, even children covered by private plans may suffer from poor health, and company costs are inflated by improper prescription practices.

The present study demonstrates that preventive health can be provided at modest costs and achieve significant savings. Mothers accepted well-baby services and had no complaints about reduced medications. The advantages to the employer of preventive health care are clear. In 1989, two more mines initiated services. If the model is replicated throughout the mining community, more than 200 000 children in the most remote locations in Peru will benefit. ☐

Acknowledgments

This work was supported by Contract No. AID/DPE 3035-C-5047-00 between John Short & Associates and the US Agency for International Development under the auspices of the Technical Information on Population for the Private Sector (TIPPS) Project.

This paper is dedicated to the memory of Bruno Lesevic, MA. The authors wish to thank the Milpo Mining Company for offering unlimited access to records and facilities. They also acknowledge the participation of Carlos Aramburu, Miriam Rojo, and Salvador Baldizon. An earlier version of this paper was presented by Foreit, Baldizon, and Winterhalter at the 117th Annual Meeting of the American Public Health Association, October 1989, Chicago, Ill.

References

1. Zschock DK, ed. *Health Care in Peru, Resources and Policy.* Boulder, Colo: Westview Press; 1988.
2. Berkow R, Talbott JH, eds. *The Merck Manual of Diagnosis and Therapy.* 13th ed. Rahway, NJ: Merck Sharp & Dohme Research Laboratories; 1977.
3. Baker CE, publisher. *Physicians' Desk Reference.* 34th ed. Oradell, NJ: Medical Economics Company, Litton Industries, Inc; 1980.
4. Panamericana de Libros de Medicina, SA. *Diccionario de Especialidades Farmaceuticas.* 1st ed., Peru. Bogota, Colombia; 1982.
5. World Health Organization. Treatment of children with acute respiratory infection: simplified models for therapeutic decisions. *Personnel Training Modules,* WHO/CDC, ECP/ARI. Geneva, Switzerland; 1985.

Measles Vaccination Coverage among Latino Children Aged 12 to 59 Months in Los Angeles County: A Household Survey

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ABSTRACT

A high rate of measles transmission has occurred in Los Angeles County since 1987. To determine measles vaccination coverage among Latino preschoolers, the group at highest risk of measles during this period, a cluster survey was conducted among Latino households with children aged 12 to 59 months. Of 279 children enrolled, 240 (86%, 95% confidence interval = 81% to 91%) reportedly had received the measles vaccine. Measles vaccination coverage among Latino preschoolers in Los Angeles County is insufficient to halt measles transmission within this subpopulation. (*Am J Public Health.* 1991;81:1057–1059)

Introduction

The incidence of measles in Los Angeles County (LAC) was less than 2.0 cases per 100 000 person-years from 1985 to 1987, but measles incidence increased to 7.3 in 1988[1] and 15.3 in 1989[2] (Table 1), with Latinos experiencing the highest rate among all ethnic groups during both years. Of the 1366 probable and confirmed measles cases occurring between January 1988 and June 1989, 601 (44%) were Latino preschoolers, the group at highest risk during this period.[2]

To promote early immunization and to reduce the number of children susceptible to measles, the LAC Department of Health Services lowered the recommended age of measles-mumps-rubella (MMR) vaccination from 15 to 12 months of age in July 1988.[3] During 1989, the same agency conducted a household cluster survey among Latino preschoolers to assess vaccination coverage.

Methods

The household survey was performed with a cluster sampling technique similar to that commonly used by the World Health Organization's Expanded Programme on Immunization (EPI).[4,5] The sampling frame included the 344 census tracts in LAC consisting of 50% or more Latino residents; two thirds of LAC Latinos reside within these census tracts. Census tracts were determined to be 50% or more Latino based on the 1980 census, adjusted for reported births and deaths through 1988.

Thirty census tracts were systematically selected from the sampling frame with probability proportionate to size. After random number tables and coin flips were used to select a street and initial household within each census tract, households were visited consecutively until seven eligible households were included in the survey. Households were not revisited if no one responded to a knock on the door.

Household inclusion criteria were permanent residence of a child aged 12 to 59 months who had at least one Latino parent, and presence of a respondent at least 16 years of age who was willing to participate. Latino was self-defined by the respondents and was generally understood to mean of Latin American descent. Possession of a written vaccination record

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This paper was submitted to the journal August 24, 1990, and accepted with revisions April 25, 1991.

TABLE 1—Measles Incidence and Case Fatality Rates in Los Angeles County,[a] 1985 to 1989

	Measles			
Year	Cases	Incidence[b]	Deaths	Case Fatality Rate (%)
1985	44	0.60	0	0
1986	40	0.53	0	0
1987	126	1.68	0	0
1988	570	7.29	2	0.35
1989	1196	15.28	14	1.17

[a] Excluding Pasadena and Long Beach cities.
[b] Cases per 100 000 person-years.
Source: Communicable Disease Control Programs: Communicable disease morbidity reports, 1985–1989. County of Los Angeles Department of Health Services, Public Health Programs and Services.

was not required but was used if available. All eligible children in the household were included in the survey.

A standard interview form was used to obtain family information and vaccination history of the child(ren). A child was considered vaccinated against measles if the child had received either MMR or monovalent measles vaccine from any source on or after the first birthday. Interviewers were bilingual (English/Spanish) and conducted interviews between 9 AM and 5 PM on weekdays, from July 18 through September 13, 1989.

Age of vaccination was calculated from the birth and vaccination dates of children with written vaccination records. Confidence intervals (CIs) for the vaccination coverage point estimates were calculated using the method described by Lemeshow and Stroh.[6] Statistical significance was assessed with Mantel-Haensel χ^2 statistics, but these CIs may be too narrow because the survey did not employ a simple random sample.

Results

Of the 1684 households visited during the survey, an adult was present at 909 (54%), 676 of which were Latino. Of the 273 Latino households with eligible children, 209 (77%) agreed to participate. Fifty-one (80%) of the 64 refusals to participate were from secondary caretakers, who denied knowledge of vaccination history. The survey included 279 children, and the child's mother was the respondent for 221 (79%).

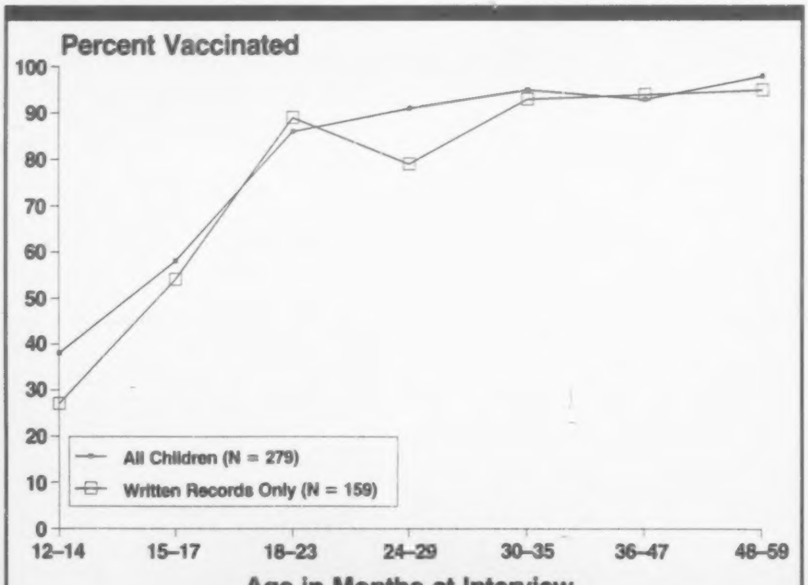

FIGURE 1—Proportion of Latino Children Residing in Predominantly Latino Census Tracts in Los Angeles County Who Were Vaccinated Against Measles, by Age Group, 1989

Respondents reported that 240 (86%; 95% CI = 81%–91%) of the children had received measles vaccine. Of the 201 children aged 24 to 59 months, 190 (95%) reportedly were vaccinated, compared with 50 (64%) of the 78 children aged 12 to 23 months (odds ratio [OR] = 9.7; 95% CI = 4.5–20.7) (Figure 1).

Written vaccine records were available for 159 (57%) of the children. Measles vaccine had been received by 131 (82%; 95% CI = 75% to 90%) of these children, a smaller proportion than that reported for children without written records (90%; 95% CI = 86–96%) (Figure 1). Mean age of measles vaccination was 19.5 months for the 131 vaccinated children, and mean age at interview was 21.5 months for the 28 unvaccinated children. Of the 46 children aged 15 to 29 months, 22 (48%) were vaccinated before 16 months of age, compared with 33 (34%) of the 98 children aged 30 to 59 months (OR = 1.8; 95% CI = 0.9–3.7) (Figure 2). Of the 112 children who were at least 2 years of age, 76 (68%) had been vaccinated before their second birthday.

Discussion

Effective measles vaccination programs aim to vaccinate a large enough proportion of susceptible persons (typically 92% to 96%) to attain herd immunity.[7] However, in nonrandomly mixed populations, such as the population of LAC, the number of susceptible persons and the frequency of their contact are more important in limiting transmission than the proportion of immune individuals within the whole population.[8] Because measles transmission was primarily focused in preschool children during 1989 and measles vaccination coverage was less than 92% to 96% among Latino preschoolers, endemic transmission will likely continue among Latinos in LAC until the number of susceptible preschool children decreases substantially, either through natural infection or vaccination.

Measles vaccination was delayed on the average until about 20 months of age among Latino children in this survey, and more than two thirds of the nonvaccinated children were younger than 24 months of age. These findings highlight the importance of delayed vaccination in the estab-

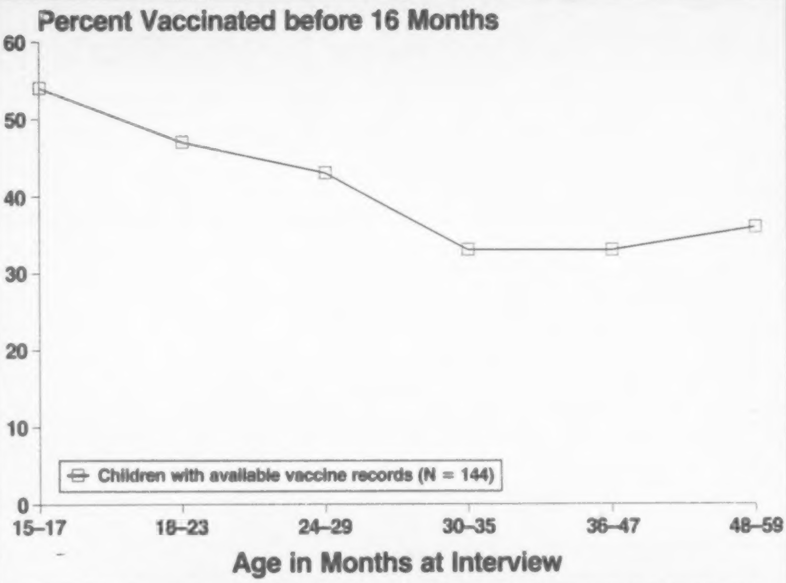

FIGURE 2—Proportion of Latino Children Residing in Predominantly Latino Census Tracts in Los Angeles County and for Whom Vaccine Records Were Available, Who Were Vaccinated Against Measles Before 16 Months of Age, by Age Group, 1989

lishment of a pool of children susceptible to measles. Fortunately, since the July 1988 change of recommended age of MMR vaccination from 15 to 12 months, the proportion of children vaccinated before 16 months of age has steadily increased (Figure 2).

The higher coverage reported among children without written records may reflect a tendency for the caretakers of these children to report vaccination for unvaccinated children. Inclusion of these children may have resulted in an overestimation of vaccination coverage. Also, children with mothers working outside the household may have been undersampled, since households were not revisited if no one answered the door or a knowledgeable respondent was not present. The effect of this potential bias on the coverage estimate is unknown.

Our sample variation from EPI—selection of seven households instead of seven children per cluster—had a negligible effect on the coverage estimate. An analysis that excluded the children who would not have been included in an EPI sample revealed a less than 1% difference in vaccination coverage. However, nonprobability sampling at the second stage, an inherent problem of the EPI-style survey, may have had some unknown effect on the vaccination coverage estimate.

Measles continues to affect primarily preschoolers in LAC, and the new two-dose MMR vaccination schedules recommended by the American Academy of Pediatrics[9] and the Immunization Practices Advisory Committee[10] will not reduce measles incidence among these children. Since about 95% of children receiving a second dose of MMR are already immune to measles,[11] public health efforts should focus on protection of unvaccinated preschoolers in areas where these children constitute the main risk group and resources are limited. □

Acknowledgments

The authors wish to thank Maria Becerra-Cruz, Mary Bellafiore, Patricia Garcia, Rosemary Maccani, and Evelyn Vega for their assistance in data collection.

The data in this paper were presented at the 117th Annual Meeting of the American Public Health Association in Chicago, October 22–26, 1989, in the Epidemiology Section Late Breaker session.

References

1. Communicable Disease Control Programs. *Communicable Disease Morbidity Report, County of Los Angeles, 1988.* Los Angeles, Calif: Los Angeles County Department of Health Services, Public Health Programs and Services; 1990.
2. Communicable Disease Control Programs. *Communicable Disease Morbidity Report, County of Los Angeles, 1989.* Los Angeles, Calif: Los Angeles County Department of Health Services, Public Health Programs and Services; 1991.
3. County of Los Angeles Department of Health Services. Measles outbreak and change in MMR immunization age. *Public Health Lett.* 1988;10:25–26.
4. Henderson RH, Sundaresan T. Cluster sampling to assess immunization coverage: a review of experience with a simplified sampling method. *Bull World Health Organ.* 1982;60:253–260.
5. Rothenberg RB, Lobanov A, Singh KB, Stroh G Jr. Observations on the application of EPI cluster survey methods for estimating disease incidence. *Bull World Health Organ.* 1985;63:93–99.
6. Lemeshow S, Stroh G Jr. *Sampling Techniques for Evaluating Health Parameters in Developing Countries.* Board on Science and Technology for International Development, National Research Council. Washington, DC: National Academy Press; 1988.
7. Anderson RM, May RM. Vaccination and herd immunity to infectious diseases. *Nature.* 1985;318:323–329.
8. Fox JP, Elveback L, Scott W, Gatewood L, Ackerman E. Herd immunity: basic concept and relevance to public health immunization practices. *J Epidemiol.* 1971;94:179–189.
9. American Academy of Pediatrics Committee on Infectious Diseases. Measles: reassessment of the current immunization policy. *Pediatrics.* 1989;84:1110–1113.
10. Centers for Disease Control. Measles prevention: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR.* 1989;38(suppl):1–18.
11. Benenson AS, ed. *Control of Communicable Diseases in Man.* Washington, DC: American Public Health Association; 1990.

Low-Level Air Pollution and Upper Respiratory Infections in Children

Jouni J. K. Jaakkola, MD, Mikko Paunio, MD, Martti Virtanen, MD, and Olli P. Heinonen, MD, DSc

ABSTRACT

Effects of low-level air pollution were studied in one polluted city and two reference cities in northern Finland by comparing the frequency of upper respiratory infections over a 12-month period in 1982 as reported by parents of children ages 14 through 18 months (n = 679) and 6 years (n = 759). A similar comparison was carried out between children living in the more polluted and less polluted areas of the polluted city. The annual mean and the greatest half-hour concentrations of sulfur dioxide (23 and 807 μg/m^3), particulates (31 and 291 μg/m^3), nitrogen oxides (15 and 160 μg/m^3), and hydrogen sulfide (2 and 177 μg/m^3) in the polluted city were mainly due to industrial sources. In the reference cities, air pollution was produced mainly by traffic and heating. Adjusted odds ratios (OR) for one or more upper respiratory infections of residents in the polluted city vs those in the reference cities were 2.0 (95% confidence interval [CI] = 1.3–3.2) in the younger age group and 1.6 (95% CI = 1.1–2.1) in the older age group. Within the polluted city, OR calculated for living in more vs less polluted areas were 2.0 (95% CI = 1.0–4.0) in the younger and 1.6 (95% CI = 1.0–2.7) in the older children. The present results suggests that, for children, air pollution can be hazardous in concentrations lower than those recorded in earlier studies from Britain and central Europe. The synergistic effect of sulfur dioxide, particulates, nitrogen oxides, hydrogen sulfide, and other pollutants may be a contributing factor. (*Am J Public Health.* 1991;81:1060–1063)

Introduction

Several studies in the 1960s showed that children living in areas with sulfur dioxide (SO_2) and particulate levels above 100 μg/m^3 had an increased occurrence of respiratory symptoms, upper and lower respiratory tract illnesses, and lower levels of pulmonary function than children living in less polluted areas.[1–5] The evidence is controversial on the effects of air pollution at levels lower than 100 μg/m^3.[6–9] However, a recent study from Israel reported a significantly higher prevalence of respiratory symptoms and conditions in second- and fifth-grade children living in an area of Israel with an annual average of only 30 μg/m^3 SO_2 than in children of a less polluted area.[9]

We evaluated the effect of low-level air pollution by comparing the frequency of upper respiratory infections in infants and preschool children in Oulu (polluted city), the northernmost industrial city in Finland, and in two cities with little industrial ambient air pollution, Vaasa (southern reference city) and Rovaniemi (northern reference city); see Figure 1.

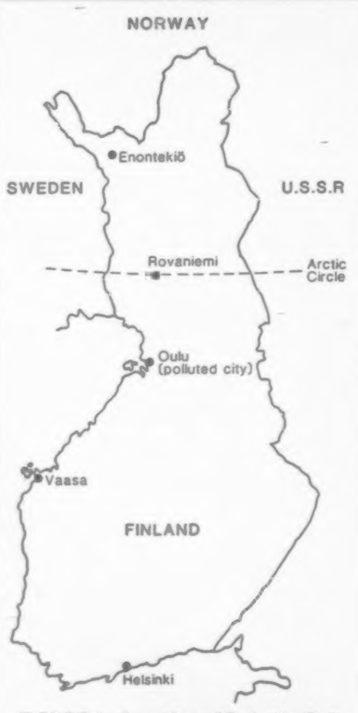

FIGURE 1—Location of Oulu, the Polluted City, and Vaasa and Rovaniemi, the Reference Cities.

Methods

Oulu (population 96 000) is located on the Baltic coast. Vaasa (coastal, population 54 000) and Rovaniemi (inland, population 31 000) are the closest southern and northern cities with an approximately similar level of urbanization but with little industrial air pollution in the downtown areas.

Four main pollution sources encircle the town center of Oulu (Figure 2). A sulfite cellulose pulp mill including a power station, northwest of the center, is the biggest source of sulfur dioxide (SO_2) pollution (2500 tonnes/y). The main source of nitrogen oxides (NO_x), a chemical plant producing acids and fertilizers, lies to the northeast. To the southwest a sulfate cellulose pulp mill, which is a source of hydrogen sulfide (H_2S) and methyl mercaptan (CH_3SH), causes odor disturbance. The heating plant of Oulu University Hospital, in the southeast, is an important source of SO_2 and particulates.

All cities monitor SO_2 and particulate concentrations. In the polluted city, H_2S and NO_x concentrations have also been measured since 1983. The measured concentrations from 1982 to 1984 are summarized in Table 1.

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This paper was submitted to the journal February 6, 1990, and accepted with revisions April 2, 1991.

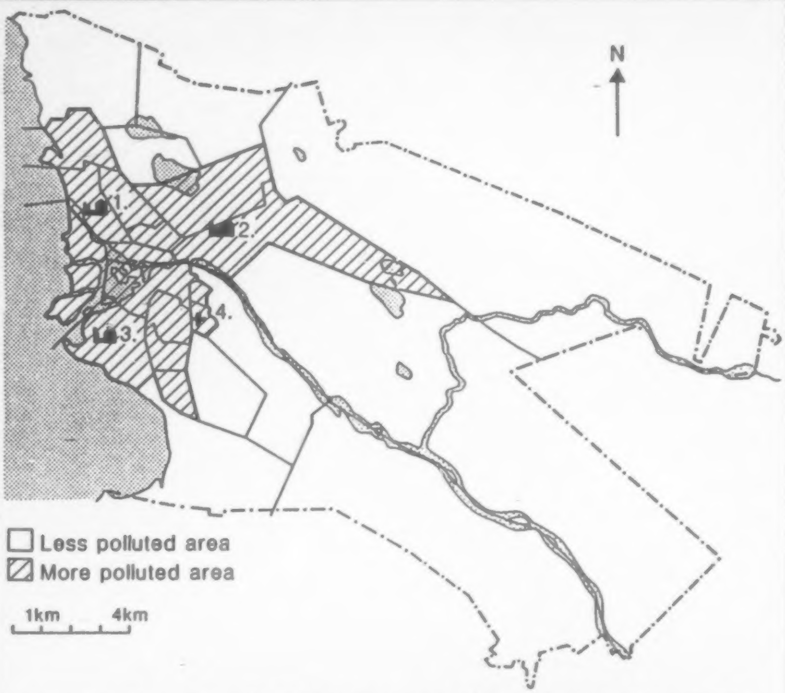

FIGURE 2—The Polluted City Divided Into Areas of More and Less Pollution: Building 1, Sulfite Cellulose Pulp Mill and a Power Plant; Building 2, Chemical Plant; Building 3, Sulfate Cellulose Pulp Mill; and Building 4, University Hospital.

Although the long-term mean levels of SO_2 were only slightly greater in the polluted city than in the reference cities, there were more frequent periods of short-term, high-level exposure to SO_2 due to downtown industrial sources. The population of the polluted city was exposed to considerably greater levels of H_2S, CH_3SH, and other malodorous sulfur compounds and nitrogen oxides.

The polluted city was divided into areas of more and less pollution according to the dispersal models of SO_2 made by the Finnish Meteorologic Institute. The more polluted area included all postal districts with an annual mean concentration of SO_2 ≥ 15 $\mu g/m^3$ and two postal districts with NO_x produced by the chemical plant but with SO_2 concentrations <15 $\mu g/m^3$. The less polluted area included those districts with an annual mean SO_2 concentration <15 $\mu g/m^3$ together with low levels of NO_x (Figure 2).

TABLE 1—Air Pollution in the Polluted City and Southern and Northern Reference Cities, 1982–1984

Pollutant ($\mu g/m^3$)	Polluted City	Southern Reference City	Northern Reference City
Sulfur dioxide			
Annual mean	13–23	9–19	17–18
Daily maximum	57–83	37–56	53
Half-hour maximum	360–807	120–190	80
Particulates			
Annual mean	31	20–33	17–28
Daily maximum	73	74–198	100–103
Half-hour maximum	291	—	—
Nitrogen oxides[a]			
Annual mean	15	—	—
Daily maximum	48	—	—
Half-hour maximum	161	—	—
Hydrogen sulfide[a]			
Annual mean	2.3	—	—
Daily maximum	42.3	—	—
Half-hour maximum	177.0	—	—

[a] Concentrations in reference cities estimated to be low due to lack of sources.

The data on health status were collected during the first two months of a mumps-measles-rubella vaccination program in November and December 1982. The total number of children ages 14 to 18 months was 679 (364, 190, and 125 from Oulu, Vaasa, and Rovaniemi, respectively) and the total of children age 6 years was 759 (310, 305, and 144, respectively).

During the vaccination visit, a nurse checked individual health records and interviewed the parents as to name, age, sex, the number of upper respiratory infections with fever (common cold, otitis media) during the previous 12 months, allergic diseases confirmed by physician (obstructive bronchitis or asthma, allergic rhinitis or conjunctivitis, and atopic dermatitis), passive smoking at home, and the number of daily contacts with other children at home and day care. The socioeconomic status of parents was obtained from the 1980 national census but was available only for older children.

The adjusted odds ratios for upper respiratory infections for those living in the more polluted vs the less polluted area were estimated in logistic regression analyses (see Appendix). The outcome was dichotomized (no infection = 0, one or more infections = 1). Similarly, other independent variables in the model were dichotomized. A child having any of the allergic diseases was considered atopic (no = 0, yes = 1). The main effects model was fitted by taking the determinant of interest (exposure) and all the other potential determinants of upper respiratory infections in the model. Meaningful interaction terms were considered according to their statistical significance ($\beta/SE[\beta] < 1.96$).

Results

In the polluted city, both younger and older children had had more upper respiratory infections during the previous 12 months than children in the reference cities (Table 2). Within the polluted city the children in the more polluted area had had more infections than children in the less polluted area. The distributions of potential confounders in the polluted and reference cities are presented in Table 3.

TABLE 2—The Occurrence of Upper Respiratory Infections in the Polluted vs Reference Cities

No. of infections during previous 12 months	Polluted city			Southern reference city	Northern reference city	Total	Unadjusted Odds Ratio[a] (95% confidence interval[b])
	More polluted area n (%)	Less polluted area n (%)	Total n (%)	n (%)	n (%)	n (%)	
14- to 18-month old children							
0	15 (8.0)	25 (14.2)	40 (11.0)	33 (17.4)	32 (25.6)	65 (15.5)	1.0
1–4	134 (71.3)	125 (71.0)	259 (71.1)	137 (72.1)	74 (59.2)	211 (69.2)	1.9 (1.5, 5.1)
≥5	39 (20.7)	26 (14.8)	65 (17.9)	20 (10.5)	19 (15.2)	39 (15.3)	2.7 (1.6, 4.8)
Total	188	176	364	190	125	315	
6-year-old children							
0	29 (21.8)	53 (29.9)	82 (26.5)	92 (30.2)	67 (46.5)	159 (31.8)	1.0
1–4	97 (72.9)	118 (66.7)	215 (69.3)	202 (66.2)	76 (52.8)	278 (65.0)	1.5 (1.2, 6.0)
≥5	7 (5.3)	6 (3.4)	13 (4.2)	11 (3.6)	1 (0.7)	12 (3.2)	2.1 (0.9, 8.2)
Total	133	177	310	305	144	449	

[a] All polluted vs reference.
[b] Miettinen's test-based method.

The only difference of note was the number of daily child contacts for 6-year-old children.

Living in the polluted city vs reference cities was a significant risk factor for upper respiratory infections in both age groups when adjusting for potential confounders in logistic regression analysis (Table 4, Appendix). The adjusted odds ratio (OR) was 2.0 (95% CI [confidence interval] = 1.3–3.2) in the younger age group and 1.6 (95% CI = 1.1–2.1) in the older age group. Living in the more vs less polluted area of the polluted city was also a significant risk factor for upper respiratory infections in younger children (Table 3). The adjusted OR was 2.0 (95% CI = 1.0–4.0), i.e., a 100% increase in the frequency of children with infections. In 6-year-old children the corresponding adjusted OR was 1.6 (95% CI = 1.0–2.7), which was nonsignificant ($P < .10$), but the number of children in this analysis was small. When the less polluted area was enlarged to include surrounding municipalities, the adjusted OR became significant, 2.0 (95% CI = 1.3–3.0). There were no significant interactions in the comparisons between the more and less polluted areas.

TABLE 3—The Occurrence of Risk Factors in the Study Populations of Polluted and Reference Cities

	Polluted city			Southern reference city	Northern reference city	Total
	More polluted area n (%)	Less polluted area n (%)	Total n (%)	n (%)	n (%)	n (%)
14- to 18-month old children						
Number of daily contacts						
More than 4	22 (11.7)	26 (14.8)	48 (13.7)	23 (12.1)	11 (8.8)	34 (10.8)
4 or fewer	166 (88.3)	150 (85.2)	316 (86.3)	167 (87.9)	114 (91.2)	281 (89.2)
Passive smoking at home						
Yes	57 (30.3)	46 (26.1)	103 (28.3)	51 (26.8)	38 (30.4)	89 (28.3)
No	131 (69.7)	130 (63.9)	261 (71.7)	139 (73.2)	87 (69.6)	226 (71.7)
Atopic disease						
Yes	25 (13.3)	29 (16.5)	54 (14.8)	18 (9.5)	16 (12.8)	34 (10.8)
No	163 (86.7)	147 (83.5)	310 (85.2)	172 (90.5)	109 (87.2)	121 (89.2)
6-year-old children						
Number of daily contacts						
More than 4	111 (83.5)	134 (75.7)	245 (79.0)	280 (91.8)	107 (74.3)	387 (86.2)
4 or fewer	22 (16.5)	43 (24.3)	65 (21.0)	25 (8.2)	37 (25.7)	62 (13.8)
Passive smoking at home						
Yes	52 (39.1)	68 (38.4)	120 (38.7)	102 (33.4)	62 (43.1)	164 (36.5)
No	81 (60.9)	109 (61.6)	190 (61.3)	203 (60.6)	82 (56.9)	285 (63.5)
Atopic disease						
Yes	37 (20.9)	29 (21.8)	66 (21.3)	48 (15.7)	31 (21.5)	79 (17.6)
No	140 (79.1)	104 (78.2)	244 (78.7)	257 (84.3)	113 (78.5)	370 (82.4)

TABLE 4—Crude (cOR) and Adjusted Odds Ratios (aOR) for Having Experienced an Upper Respiratory Infection during the Past 12 Months in Polluted vs Reference Cities and in the More vs Less Polluted Area of the Polluted City

Population	cOR	aOR	95% CI[a]
14- to 18-month-old children			
Reference cities	1.0	1.0	
Polluted city	2.1	2.0	1.3—3.2
Less polluted area	1.0	1.0	
More polluted area	1.9	2.0	1.0—4.0
6-year-old children			
Reference cities	1.0	1.0	
Polluted city	1.5	1.6	1.1—2.1
Less polluted area	1.0	1.0	
More polluted area	1.5	1.6	1.0—2.7

Adjustment was made by logistic regression analysis controlling for sex, daily child contacts, atopic disease, and passive smoking.

[a]CI = confidence interval.

Discussion

The ambient air in the index city was more polluted than in the reference cities due to the industrial sources around the town center. There were also differences in air pollution across the polluted city. However, the differences in the annual mean concentrations of SO_2 between the cities and within the polluted city were small. Moreover, in terms of international comparison, the levels in the most polluted area in the polluted city (25 $\mu g/m^3$) can still be considered low-level air pollution. However, the industrial sources in the polluted city cause higher and more frequent peaks of SO_2 than are caused by heating and traffic in the reference cities. Industrial NO_x, H_2S, and CH_3SH emissions were the main qualitative differences in comparing the polluted city with the reference cities.

We found a significant association between the occurrence of upper respiratory infections and living in an air-polluted area. This finding was consistent in both 14 to 18 month olds and 6 year olds (and combined) when comparing the polluted city with the reference cities and when comparing the more and less polluted areas within the polluted city. Most of the potential confounders were controlled for either by the study design and/or in the multivariate analysis. There was no municipal gas distribution in any of the cities studied. Passive smoking, daily child contacts, and atopic disease were included in the logistic models. Socioeconomic classification obtained from the 1980 census was available for the older age group. The differences in socioeconomic distribution between the cities were found to be small.

The data collection was blind: neither the participants nor the interviewers were aware of the purpose of the study. Thus the potential information bias caused by knowledge of children's exposure status was eliminated.

In conclusion, our results suggest that children's respiratory health can be jeopardized from exposure to air pollution in concentrations much lower than those recorded in earlier studies from Britain and central Europe. The synergistic effect of SO_2, particulates, NO_x, H_2S, and other pollutants may be a contributing factor. □

References

1. Toyama T. Air pollution and its health effect in Japan. *Arch Environ Health*. 1964;8:161–181.
2. Douglas JWB, Waller RE. Air pollution and respiratory infections in children. Br J Prev Soc Med 1966;20:1–8.
3. Lunn JE, Knowelden J, Handyside AJ. Patterns of respiratory illness in Sheffield infant schoolchildren. *Br J Prev Soc Med.* 1967; 21:7–16.
4. Biersteker K, van Leeuwen P. Air pollution peak flow rates of school children in two districts of Rotterdam. *Arch Environ Health.* 1970;20:382–384.
5. Lunn JE, Knowelden J, Roe JW. Patterns of respiratory illness in Sheffield junior schoolchildren: a follow-up study. *Br J Prev Soc Med.* 1970;24:223–228.
6. Commission of the European Communities Environmental Research Programme. Report on the EC epidemiological survey on the relationship between air pollution and respiratory health in primary school children. Brussels; 1983.
7. Melia RWJ, Florey C du V, Chinn S. Respiratory illness in British school children and atmospheric smoke and sulphur dioxide 1973–7: II, Longitudinal findings. *J Epidemiol Community Health.* 1981;35:168–173.
8. Love GJ, Lan S-P, Shy CM, Struba RJ. The incidence and severity of acute respiratory illness in families exposed to different levels of air pollution, New York Metropolitan Area, 1971–1972. *Arch Environ Health.* 1981;36:66–74.
9. Goren AI, Hellmann S. Prevalence of respiratory symptoms and diseases in schoolchildren living in a polluted and in a low polluted area in Israel. *Environ Res.* 1988;45:28–37.

APPENDIX—Regression Coefficients Produced by PROBIT Procedure of SPSSX

Risk factor	Polluted vs reference city: Younger children β (SE)	Polluted vs reference city: Older children β (SE)	Polluted vs reference area[a]: Younger children β (SE)	Polluted vs reference area[a]: Older children β (SE)
Living in polluted city or area	0.357 (0.110)	0.221 (0.082)	0.339 (0.174)	0.256 (0.139)
More than 4 daily child contacts	0.423 (0.306)	0.121 (0.099)	0.476 (0.522)	−0.289 (0.161)
Atopic disease	0.224 (0.187)	0.140 (0.105)	0.152 (0.254)	0.073 (0.163)
Passive smoking	0.114 (0.124)	−0.140 (0.081)	−0.058 (0.185)	−0.084 (0.133)
Male sex	−0.131 (0.108)	0.025 (0.079)	−0.115 (0.170)	0.109 (0.131)
Intercept	5.674	5.217	5.926	5.595

Note: All the variables are coded yes = 1 and no = 0.

The final equation for polluted vs reference city, younger children is:

logit [pr(INFECTION = 1 /POLLUTED, CONTACTS, ATOPIC, PASSIVE, SEX)] = Intercept + 0.357(POLLUTED) + 0.423(CONTACTS) + 0.224 (ATOPIC) + 0.114(PASSIVE) − 0.131(SEX)

The procedure calculates the estimates from LOGIT model:

$$\{LOG[p/(1 - p)]\}/2 + 5 = Intercept + \beta X$$

This means that aOR can be calculated:

$$aOR = e^{2*\beta}$$

and the 95% confidence limit is

$$e^{2*\beta \pm 2*1.96*SE(\beta)}$$

[a]Restricted to the residents of the polluted city.

Decline in the Use of Illicit Drugs by High School Students in New York State: A Comparison with National Data

Denise B. Kandel, PhD, and Mark Davies, MPH

ABSTRACT

A school-based epidemiological survey of the use of drugs was carried out in Spring 1988 among 7,611 adolescents representative of students in grades 7 through 12 in New York State public and private schools. This survey provides the most recent data on the use of crack in that population. Comparison with a statewide survey carried out five years earlier reveals a large decline in the use of illicit drugs in that period. This decline appears to be stronger than the decline reported for national samples. (*Am J Public Health.* 1991;81:1064–1067)

Introduction

National epidemiological surveys[1,2] document a decrease in the use of illicit drugs starting in 1980 for marijuana and in the mid-1980s for cocaine. We report results from a 1988 survey of high school students in New York State (NYS) which reveals large declines in the use of illicit drugs over five years. The decline appears to be stronger in the Northeast than in the United States as a whole and sharper in NYS than in the entire Northeast.

Methods

A statewide epidemiological survey of the use of alcoholic beverages, cigarettes, marijuana, cocaine, crack, and other illicit drugs was conducted among 7,611 students in grades 7 through 12 in NYS in Spring 1988.

The two-stage random sample represents junior and senior high school students attending NYS public and private schools. A stratified sample of 54 schools and two homerooms from each grade per school was selected. The four stratification criteria for school selection were: 1) geographical area; 2) proportion White enrollment; 3) public versus private; 4) size of enrollment. Active or passive parental consent for the child's participation was obtained, as required by individual schools. A school in New York City was dropped because only 10 percent of parental consent forms were returned. Students answered anonymous self-administered structured questionnaires in classrooms (84 percent completion rate). The sample was weighted to reflect the variable probabilities of selection of schools and homerooms and the grade-specific absentee rate in each school; 95 percent confidence intervals were calculated using empirical standard errors from 10 random subsamples drawn from the total sample. For grade-specific rates, the standard error was estimated by multiplying the standard error, assuming simple random sampling, by the average design effect (1.53).

Results

Prevalence of Student Drug Use

The rates of use follow patterns reported by others,[4,5] but probably underestimate drug use among adolescents in the population, since school absentees and dropouts were not included (Table 1).[6-9]

Trends Over Time

In 1983, the NYS Division of Substance Abuse Services (DSAS) surveyed a representative sample of students in grades 7 to 12 from 206 public and private schools selected from a stratified sample of health service areas.[3] Structured self-administered questionnaires were given in classrooms. (Completion rates are not provided.).

Comparison of rates in 1983 and 1988 reveals sizable declines of at least 50 percent over five years in the use of almost every illicit drug, but no change in alcohol use (Table 1).

Comparison with National Data

The declines in NYS schools are striking and parallel those reported nationally for high school seniors in the Monitoring the Future (MF) study by Johnston, *et al.*[1] and for adolescents ages 12 to 17 in the population reported in the National Household Survey (NHS)[2] (Table 2). [Lower rates are reported in the household (NHS) than the school (NYS) survey,[10] and may reflect in part differences in mode of survey administration.] This decline may be greater in NYS and the Northeast than nationally. Over two years, the prevalence of cocaine use reported by high school seniors declined by 43 percent in the Northeast (23 percent in

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TABLE 1—Lifetime Prevalence Rates of the Use of Various Drugs among 7th through 12th Grade Students in New York State in 1988 and 1983 and by Grade in 1988 (95% Confidence Intervals)

	Alcohol % (CI)	Cigarettes % (CI)	Marijuana % (CI)	Stimulants % (CI)	Inhalants % (CI)	Any Cocaine % (CI)	Crack % (CI)	Psychedelics % (CI)	Sedatives % (CI)	Tranquilizers % (CI)	Heroin % (CI)	N
All 7th–12th Graders												
NYS 1988	84.4 (1.5)	57.7 (2.2)	27.5 (1.3)	11.1 (0.8)	6.3 (0.6)	5.9 (0.5)	1.9 (0.4)	4.8 (0.7)	4.1 (0.7)	3.6 (0.4)	1.4 (0.4)	(7,611)
NYS 1983[a]	83	N/A	46	25	22	14	N/A	10	12	12	3	(27,414)
Grade-Specific NYS 1988												
7th grade	66.1 (4.2)	36.0 (4.2)	3.7 (1.7)	1.7 (1.1)	2.8 (1.5)	1.5 (1.1)	.8 (0.8)	1.1 (0.9)	.9 (0.8)	1.0 (0.9)	1.1 (0.9)	(1,210)
8th grade	78.2 (3.7)	51.9 (4.5)	12.2 (3.0)	2.8 (1.5)	4.4 (1.9)	1.8 (1.2)	.7 (0.8)	2.2 (1.3)	1.6 (1.1)	2.0 (1.3)	.6 (0.7)	(1,149)
9th grade	84.5 (3.0)	58.6 (4.0)	24.3 (3.5)	11.2 (2.6)	6.0 (2.0)	4.2 (1.6)	2.3 (1.2)	4.6 (1.7)	4.4 (1.7)	4.8 (1.8)	2.2 (1.2)	(1,367)
10th grade	88.7 (2.6)	61.1 (4.0)	30.1 (3.8)	14.1 (2.9)	6.7 (2.1)	5.4 (1.9)	1.9 (1.1)	5.4 (1.9)	4.7 (1.8)	3.8 (1.6)	1.1 (0.9)	(1,361)
11th grade	93.0 (2.1)	66.4 (3.9)	42.8 (4.1)	16.8 (3.1)	8.1 (2.3)	8.5 (2.3)	2.1 (1.2)	5.7 (1.9)	5.6 (1.9)	4.2 (1.7)	1.6 (1.0)	(1,348)
12th grade	94.7 (2.0)	70.9 (4.1)	50.9 (4.5)	18.8 (3.5)	9.7 (2.7)	14.0 (3.1)	3.3 (1.6)	9.6 (2.7)	7.4 (2.4)	5.5 (2.1)	1.8 (1.2)	(1,156)

N/A = Not ascertained. Cigarettes use ascertained for last month not lifetime.
[a]Source: DSAS, 1984.[3] Confidence intervals not available.

1986 versus 13 percent in 1988[1,11]), compared to 28 percent nationally (17 percent versus 12 percent). We compared the changes in rates in NYS over comparable periods in MF (1983–1988) and NHS (1982–88) (Table 3). For NYS, we calculated odds ratios for 11th–12th graders, since separate 1983 data for the 12th grade were not available.

The odds ratios are consistently and significantly higher in NYS than in the nation, except for sedatives and tranquilizers. In MF, the ratios are slightly higher in the Northeast than in the total sample and significantly different for marijuana, cocaine and stimulants.

Discussion and Conclusion

The decline in drug use appears to be sharper in areas where rates were initially the highest. This differential decline may be explained by changes in the age structure of the US population, peer influence processes, and the relative commitment of youth to substance use in areas with high compared to low use prevalence. Substance use is most prevalent in the late teens and early twenties.[2,5] The downward trend in illicit drug use prevalence from 1980 to 1988 parallels the decline in the ratio of youths (aged 15–24) to the parental generation (aged 34–44) between

TABLE 2—Comparisons of Prevalence Rates of Lifetime Drug Use among High School Seniors in Monitoring the Future in 1988 and 1983 Nationally and in the Northeast and among 12- to 17-Year-Olds in 1988 and 1982 in the National Household Survey

	Alcohol % (CI)	Cigarettes % (CI)	Marijuana % (CI)	Stimulants % (CI)	Inhalants % (CI)	Any Cocaine % (CI)	Crack % (CI)	Psychedelics % (CI)	Sedatives % (CI)	Tranquilizers % (CI)	Heroin % (CI)	N
Monitoring the Future												
12th Grade												
88 National[a]	92.0 (1.5)	66.4 (1.7)	47.2 (2.1)	19.8 (1.5)	16.7 (1.3)	12.1 (1.2)	4.8 (0.7)	8.9 (1.0)	7.8 (1.0)	9.4 (1.1)	1.1 (0.3)	(16,796)
83 National[b]	92.6 (1.3)	70.6 (1.5)	57.0 (2.1)	26.9 (1.5)	18.2 (1.2)	16.2 (1.4)	N/A	11.9 (1.0)	14.4 (1.3)	13.3 (1.3)	1.2 (0.3)	(16,300)
88 Northeast[a]	93.9	66.6	49.6	16.5	15.3	13.2	3.8	9.3	7.7	8.1	1.0	(3,346)
83 Northeast[b]	95.4	72.9	63.7	26.9	13.0	20.5	N/A	14.0	12.4	12.3	1.1	(4,056)
National Household Survey												
12–17 year olds												
88 National[d]	50.2 (5.7)	42.3 (4.2)	17.4 (4.1)	4.2 (2.1)	8.8 (2.1)	3.4 (1.7)	.9 (0.8)	3.5 (1.5)	2.4 (1.8)	2.0 (1.5)	.6 (0.5)	(3,095)
82 National[c]	65.2 (3.0)	49.5 (3.1)	26.7 (2.7)	6.7 (1.6)	N/A	6.5 (1.6)	N/A	5.2 (1.4)	5.8 (1.5)	4.9 (1.4)	<.5	(1,581)
88 Northeast[d]			17.3									(566)
82 Northeast[c]			31.0									(337)

N/A = Not ascertained.
Sources: [a]Monitoring the Future 1988[1]—12th graders. Standard errors not available for Northeast.
[b]Monitoring the Future 1983[10]—12th graders. Standard errors not available for Northeast.
[c]National Household Survey 1982[11]—12–17 year olds. Northeast data not presented for age specific groups, except marijuana; standard errors not available.
[d]National Household Survey 1988[2]—12–17 year olds. Northeast data not presented for age specific groups, except marijuana; standard errors not available.

TABLE 3—Odds Ratios for Changes in Rates of Lifetime Drug Use Over a 5-Year Period among Adolescents in National Samples, the Northeast and New York State from 1983 (or 1982) to 1988 Odds Ratios

	Any Alcohol % (S.E.)	Cigarettes % (S.E.)	Marijuana % (S.E.)	Stimulants % (S.E.)	Inhalants % (S.E.)	Any Cocaine % (S.E.)	Psychedelics % (S.E.)	Sedatives % (S.E.)	Tranquilizers % (S.E.)
Monitoring the Future[a]									
12th graders (1983–1988)									
National	1.1 (.05)	1.2 (.03)	1.5 (.03)	1.5 (.09)	0.8 (.02)	1.4 (.04)	1.4 (.05)	2.0 (.07)	1.5 (.05)
Northeast	1.4 (.15)	1.4 (.07)	1.8 (.09)	1.9 (.11)	0.8 (.05)	1.7 (.11)	1.6 (.12)	1.7 (.14)	1.3 (.11)
National Household Survey[b]									
(12–17 years old) (1982–1988)									
National	1.9 (.12)	1.3 (.08)	1.7 (.13)	1.6 (.22)	N/A	2.0 (.28)	1.5 (.22)	2.5 (.40)	2.5 (.43)
Northeast	N/A	N/A	2.2 (.36)	N/A	N/A	N/A	N/A	N/A	N/A
New York State[c] (1983–1988)									
7th–12th graders	.9 (.03)	N/A	2.2 (.06)	2.7 (.11)	4.2 (.21)	2.6 (.13)	2.2 (.13)	3.2 (.19)	3.6 (.23)
11th–12th graders	N/A	N/A	2.1 (.10)	2.7 (.15)	2.3 (.18)	2.1 (.15)	2.3 (.19)	2.2 (.19)	1.2 (.12)

N/A = Data not available in published reports.
Sources: [a]Johnston, O'Malley, and Bachman.[10,1]
[b]Miller et al,[11]; NIDA.[2]
[c]DSAS[3]; present study.

the early and late 1980s, from 1.7 to 1.1. (The upward trend in prevalence from 1960 to 1980 paralled the upward trend in the ratio of youths to adults.) Fewer members in one's age cohort and smaller relative cohort size will reduce opportunities for social interactions with one's peers, a most important factor in drug use initiation,[14] and increase social control by the older generation.[15,16] Perceived risks and disapproval associated with illicit drug use have increased.[17,18] Although changes in relative cohort sizes are almost identical in NYS and the US, the sharper decline in drug use prevalence in NYS could be accounted for by the structural factor, if changes in individual behavior depend not only on opportunities for peer interactions but on group norms and drug users' characteristics. The rate of decline in drug use may follow a reverse diffusion process characteristic of epidemics, and may accelerate as a function of the number of individuals initially exposed, especially if proscriptive norms also become more negative and individuals at risk for drug involvement are less deviant. In areas with high prevalence of drug use, less deviant youths will be drawn into drug use, mainly through peer influence. The high use areas would show more rapid decline in prevalence than areas with low prevalence, where the pool of existing and potential users would include individuals more committed to drugs.

The decrease in lifetime drug use observed among NYS students cannot be attributed to an increase in school dropout rates by drug-using adolescents, thereby leaving in schools an increasing number of non-drug users, since the dropout rate has been declining.[15]

These epidemiological data contrast with data on treated or clinical cases, which have shown dramatic increases throughout the 1980s,[20] although recent data on cocaine-related emergency room admissions show declines.[21] There may be a time-lag between initiating drug use and experiencing drug-related problems; smoked cocaine now accounts for a large part of clinical cases.

The most extreme forms of drug use may be concentrated in disorganized communities that are not well represented in school or household samples. Whether the situation in these communities has deteriorated over the last decade is not known. We may be moving toward an increasingly polarized society, in which drug involvement is another dimension, besides lower economic and social resources and higher social pathologies,[22] on which groups are divided and the gap between them is becoming greater.[22,23] There would be youths in school and mainstream society, who have decreased their drug use, and those in urban ghettos and out of school, who have increased their use.[24] Strengthening the educational system and youth's investment in their schooling and future may be a most promising way of reducing involvement in such activities as drug use, which are destructive for self and society. ☐

Acknowledgments

Work on this research was partially supported by an award from the New York State Department of Mental Hygiene, a grant from the William T. Grant Foundation and Research Scientist Award KO5 DA00081 from the National Institute on Drug Abuse. Partial support for computer costs was provided by Mental Health Clinical Research Center Grant MH30906 from NIMH to the New York State Psychiatric Institute. The research assistance of Byron Davis and Christine Schaffran is gratefully acknowledged.

References

1. Johnston LD, O'Malley PM, Bachman JG: Drug Use, Drinking, and Smoking: National Survey Results from High School, College, and Young Adult Populations. 1975–1988. Rockville, MD: NIDA, 1989.
2. National Institute on Drug Abuse: National Household Survey on Drug Abuse: Population Estimates. Rockville, MD: NIDA, 1989a.
3. New York State Division of Substance Abuse Services: Substance Use among New York State Public and Private School Students in Grades 7 through 12. New York City, NY, 1984.
4. Kandel DB, Single E, Kessler R: The epidemiology of drug use among New York State high school students: Distribution, trends and change in rates of use. *Am J Public Health.* 1976;66:43–53.
5. Kandel DB, Logan JA: Patterns of drug use from adolescence to young adulthood—I. Periods of risk for initiation, continued use, and discontinuation. *Am J Public Health.* 1984;74:660–666.
6. Clayton RR, Voss HL: Drug Use and Abuse among Student Dropouts. Technical Review on Drug Abuse and Dropouts. Rockville, MD: NIDA, 1982.
7. Mensch BS, Kandel DB: Dropping out of high school and drug involvement. *Sociol Educ.* 1988;61:95–113.
8. Kandel DB: Reaching the hard-to-reach: Illicit drug use among high school absentees. *Addict Dise.* 1975;1:465–480.
9. Kandel DB, Raveis VH, Kandel PI: Continuity in discontinuities: Adjustment in

young adulthood of former school absentees and school dropouts. *Youth Soci.* 1984; 13:325–352.

10. Johnston LD, O'Malley PM, Bachman JG: Highlights from Drugs and American High School Students 1975–1983. Rockville, MD: NIDA, 1984.
11. Miller JD, Cisin IH, Gardner-Keaton H: National Survey on Drug Abuse: Main Findings 1982. Rockville, MD: NIDA, 1983.
12. Rootman R, Smart RG: A comparison of alcohol, tobacco and drug use as determined from household and school surveys. *Drug Alcohol Dependence.* 1985;16:89–94.
13. Johnston LD, O'Malley PM, Bachman JG: National Trends in Drug Use and Related Factors among American High School Students and Young Adults, 1975–1986. Rockville, MD: NIDA, 1987.
14. Kandel DB: Developmental stages in adolescent drug involvement. *In:* Lettieri D, Sayers M, Pearson HW (eds): Theories on Drug Abuse. Rockville, MD: NIDA, 1980; 120–127.
15. Easterlin RA: Birth and Fortune: The Impact of Numbers on Personal Welfare. Chicago: University of Chicago Press, 1987.
16. Ryder NB: The cohort as a concept in the study of social change. *Am Soc Rev.* 1965; 30:843–861.
17. Bachman JG, Johnston LD, O'Malley PM, Humphrey RH: Explaining the recent decline in marijuana use: Differentiating the effects of perceived risks, disapproval, and general lifestyle factors. *J Health Soc Behav.* 1988;29:92–112.
18. Bachman GB, Johnston LD, O'Malley PM: Explaining the recent decline in cocaine use among young adults: Further evidence that perceived risks and disapproval lead to reduced drug use. *J Health Soc Behav.* 1990;31:173–184.
19. New York State Department of Education: The State of Learning. Statewide Profile of the Educational System. Albany, NY: State University of New York, 1990.
20. National Institute on Drug Abuse: Annual Data 1988. Data from the Drug Abuse Warning Network. Rockville, MD: NIDA, 1989b.
21. Office of National Drug Control Policy; Leading Drug Indicators. White Paper. Washington, DC: Executive Office of the President, 1990.
22. Wilson WJ: The Truly Disadvantaged. Chicago, IL: University of Chicago Press, 1987.
23. Son S, Model SW, Fisher GA: Polarization and progress in the Black community: Earnings and status gains for young Black males in the era of affirmative action. *Sociol Forum.* 1989;4:309–327.
24. Brunswick AF: Young Black males and substance use. *In:* Gibbs JT, Brunswick AF, Connor ME, *et al* (eds): Young, Black, and Male in America. Dover, MA: Auburn House Publishing Co., 1988;166–187.

Surveillance of Attempted Suicide among Adolescents in Oregon, 1988

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ABSTRACT

In January 1988, Oregon became the first state to require hospital-based reporting of attempted suicide (AS) in all adolescents < 18 years old. From January to December 1988, 644 cases of AS were reported (annual rate of 214 per 100 000 population, ages 10 to 17 years). We compared these 644 cases of AS with all 137 Oregon adolescents < 18 years old who committed suicide in Oregon during the 10-year period 1979 through 1988, and found that the strongest predictor of outcome was method used. (*Am J Public Health.* 1991;81:1067–1069)

Introduction

Nationally, suicide is the third leading cause of death among adolescents and young adults.[1] For each completed suicide, 30 to 200 suicides are attempted, but not completed.[2–6] Ten percent of adolescents who attempt suicide make further attempts within 1 year, and 31% do so within 2 years.[7,8]

In Oregon, suicide among teenagers is second only to motor vehicle injuries as a cause of death, accounting for 16% of deaths in Oregonians ages 13 to 19.[9,10] In 1987 the Oregon legislature mandated the reporting of all attempted suicides among persons younger than 18 years. This paper summarizes the first year (1988) of information from this surveillance system and compares the characteristics of adolescents who attempted suicide in 1988 with those who completed suicide in Oregon during the 10-year period 1979 to 1988.

Methods

Beginning January 1988, each Oregon hospital (n = 70) was required to report to the Oregon Health Division information on all adolescents younger than 18 years who attempted suicide. An attempted suicide was defined as self-inflicted injury or condition specified by the medical provider as having fatal intent, treated at a hospital or a hospital emergency department.

Information collected by the hospital included demographics, date and place of attempt, family living situation, history of previous suicide attempts, use of alcohol, results of blood alcohol testing if done, and method of attempt. This information, without name identifiers, was sent monthly to the Health Division.

For comparison, state death certificates and medical examiner's reports were used to identify all Oregon adoles-

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This paper was submitted to the journal August 27, 1990, and accepted with revisions March 6, 1991.

TABLE 1—Gender, Race, and Place of Residence of Attempted and Completed Suicide[a] among Adolescents in Oregon

Characteristic	Attempters No.	Attempters %	Completers No	Completers %	OR[b]	CI[c]
Gender						
Female	538	83.6	27	19.7	20.8	12.7–34.3
Male	106	16.4	110	80.3		
Race						
White	547	84.9	127	92.6	2.2	1.1–4.9
Other	97	15.1	10	7.4		
Residence						
Urban	128	19.8	25	18.2		
Suburban	256	39.8	47	34.3		
Rural	260	40.3	65	47.4		

[a] For attempters annual rate for 1988; for completers, average annual rate for 1979–1988.
[b] OR = odds ratio.
[c] CI = confidence interval.

TABLE 2—Age-Specific Annual Incidence Rates of Attempted and Completed Suicide among Adolescents in Oregon[a]

Age (years)	Attempters No[b]	Attempters Rate[c]	Completers No.	Completers Rate[c]	Rate ratio (A/C)[d]
10–11	6	7.2	4	0.5	14.4
12–13	72	91.6	9	1.1	83.2
14–15	248	298.3	38	4.5	66.3
16–17	309	340.6	86	9.5	35.9
Total	635	214.0	137	4.6	46.5

[a] For attempters, annual rate for 1988; for completers, average annual rate for 1979 to 1988.
[b] Age is unknown for nine attempters.
[c] Rate per 100 000 age-specific population.
[d] A = attempters; C = completers.

TABLE 3—Method Used for Attempted and Completed Suicide[a] among Adolescents in Oregon

Method	Attempters No.	Attempters %	Completers No.	Completers %
Drug overdose	486	75.5	6	4.4
Laceration or stabbing	67	10.4	0	0.0
Poisons or caustics	42	6.5	1	0.7
Hanging	10	1.6	23	16.8
Firearms	4	0.6	99	72.3
Other or unknown[b]	35	5.4	8	5.8
Total	644	100.0	137	100.0

[a] For attempters, annual rate for 1988; for completers, average annual rate for 1979–1988.
[b] Other or unknown methods include jumping, drowning, motor vehicle crash, unknown.

cents 10 to 17 years old who committed suicide in Oregon during the years 1979 to 1988. A completed suicide was defined as a person who died as a result of intentional self-inflicted injuries as determined by the medical examiner.

In this study, the term adolescent means 10- to 17-year-olds. Adolescents who attempted suicide but were not successful are termed attempters, adolescents who committed suicide are termed completers, and incidence rates are expressed per 100 000 population of 10- to 17-year-olds. Rural counties were those with less than 15 000 population; suburban were those with populations of 15 000 to 35 000; and urban were those with greater than 35 000 population.

Population data were obtained from the Bureau of the Census and the Center for Population Research and Census at Portland State University. For attempters, we used 1988 mid-year population estimates for denominators to calculate annual incidence rates. For completers, we used 1984 population estimates. Annual incidence rates were calculated for completers using 10 years of data from 1979 to 1988. This methodology was chosen after we had compared the characteristics of completers from 1979 to 1983 with those from 1984 to 1988 and found no differences by age, sex, race, or method of attempt.

Data were analyzed using Dbase, EPIINFO, and SAS. Exact confidence limits were calculated using methods based on Fisher's Exact Test.

Results

In 1988, 644 Oregon adolescents ages 10 to 17 years were reported to have attempted suicide, an annual incidence rate of 214 per 100 000. There were no attempted suicides in children younger than 10 years. Of attempters, 87.7% were aged 14 to 17 years, 83.5% were female, 84.9% were White, and 19.8% were urban residents (Table 1).

Only 9.5% (61/644) of the attempters had blood alcohol tested. Of those tested, 50.8% had detectable blood alcohol levels. The medical examiner tested 41.6% (57/137) of the completers for blood alcohol. Of completers tested, 35.1% had documented detectable blood alcohol at the time of their death.

From 1979 to 1988, 137 Oregon adolescents committed suicide, an average annual incidence rate of 4.6 per 100 000. The ratio of the average annual incidence rates of attempters to completers was 47:1. The average annual incidence rates for both attempted and completed suicide was lowest for 10 to 11 year olds and highest for 16 to 17 year olds (Table 2).

Compared with attempters, completers were more than 20 times more likely to be male and 2 times more likely to be White (Table 1). There were no statistically significant differences between attempters and completers by residence, place of attempt, day of the week, or season when the self-inflicted injury occurred.

Attempters more frequently used drug overdose as a method, while completers more frequently used firearms (Table 3). Gunshot wounds and hanging were strong predictors of fatal outcome. These two methods were used by 89.1% (122/137) of the completers compared with 2.2% (14/644) of the attempters (odds ratio [OR] = 366.0, 95% confidence interval [CI] = 162.8–844.8).

We performed a logistic regression analysis including the method, sex, race, season, residence, day of the week, and age. Choice of method was the strongest predictor of outcome ($P < .0001$). Although sex had the only other statistically significant predictor coefficient ($P = .002$), it had little effect on the predicted probability of outcome.

The univariate association of sex with outcome was primarily due to an association of sex with method. Combining data from attempters and completers showed that lethal methods were more likely to be chosen by males: 90.9% (30/33) of the hangings and 79.6% (82/103) of the shootings. In contrast, among those using stabbing or lacerations, 20.9% (14/67) were male; and among the drug overdosers, 13.0% (64/492) were male.

Discussion

In 1988, one adolescent suicide attempt with injuries severe enough to require emergency care was reported for every 467 Oregon adolescents. The ratio of attempted to completed suicide rates for those adolescents was 47:1. The overwhelming predictor of outcome was method used. Generally, adolescents who shot themselves died, while those who overdosed on drugs or lacerated themselves lived.

Collection of accurate prevalence or incidence data for attempted suicide has been hampered by a lack of central registries.[6,11] Results of limited surveys of high school students vary and have estimated that 5% to 15% of adolescents attempt suicide.[12–15] Our study showed that adolescent suicide attempts are common in Oregon and that statewide surveillance is feasible.

In this study, method and outcome were found to be inextricably linked. Thus the factors influencing one must also influence the other. Whether desired outcome determines method or the method available at the time of attempt determines outcome urgently needs clarification. For some adolescents who attempt suicide, selection of method may be paramount, and outcome will depend on method. For others, desired outcome may be decided first and selection of method will depend on that decision. Successful intervention is critically dependent on which of these two dynamics is operating. Securing firearms in a locked cabinet may not be helpful if a despondent adolescent will merely search for another lethal weapon. However, this intervention would be quite effective if the result were that an alternate, less lethal method would be used. Additional study is needed to determine factors influencing choice of method and to identify situations in which selection of method is not solely dependent on desired outcome, but is also influenced by other factors such as availability and social acceptability. ☐

Acknowledgments

The authors would like to thank Rocke Klockner, Joyce Grant-Worley, and Brent Marsden of the Oregon Health Division for their support during this study.

References

1. McGinnis JM. Suicide in America—moving up the public health agenda. *Suicide Life Threat Behav.* 1987;17:18–32.
2. Weissman M. The epidemiology of suicide attempts. *Arch Gen Psychiatry.* 1974; 30:737–746.
3. American Academy of Pediatrics, Committee on Adolescence. Suicide and suicide attempts in adolescents and young adults. *Pediatrics.* 1988;81:322–324.
4. Spirito A, Start L, Fristad M, et al. Adolescent suicide attempters hospitalized on a pediatric unit. *J Pediatr Psychol.* 1987; 12:171–189.
5. Garfinkle B, Froese A, Hood J. Suicide attempts in children and adolescents. *Am J Psychiatry.* 1982;139:1257–1261.
6. Shaw KR, Sheehan KH, Fernandez RC. Suicide in children and adolescents. *Adv Pediatr.* 1987;34:313–334.
7. Hawton K, O'Grady J, Osborn M, et al. Adolescents who take overdoses: their characteristics, problems, and contacts with helping agencies. *Br J Psychiatry.* 1982;140:118–123.
8. McIntire M, Angle C, Wikoff R, et al. Recurrent adolescent suicide behavior. *Pediatrics.* 1977;60:605–608.
9. Oregon Health Division. Reporting teenage suicide attempts in Oregon. *Commun Dis Sum.* 1988;37:1–2.
10. Oregon Health Division Report. Teenage suicide in Oregon, 1983–1985. Portland; October 1986.
11. Eisenberg L. The epidemiology of suicide in adolescents. *Pediatr Ann.* 1984;13:47–54.
12. Smith K, Crawford S. Suicidal behavior among "normal" high school students. *Suicide Life Threat Behav.* 1986;16:313–325.
13. Harkavy JM, Asnis G. Suicide attempts in adolescence: prevalence and implications. *N Engl J Med.* 1985;313:1290–1291.
14. Centers for Disease Control. Results from the national adolescent student health survey. *MMWR.* 1989;38:147–150.
15. Schwartz RH, Wirtz P. Suicide attempts by high school students. *Am J Psychiatry.* 1988;145:537.

Nurses Claim Victory in Supreme Court Decision

"Registered nurses across the country have won a major victory in the United States Supreme Court," Dr. Barbara Redman, Executive Director of the American Nurses Association (ANA), recently announced.

The court upheld an ANA-backed position allowing for separate collective bargaining units in hospitals for registered nurses.

"This decision gives nurses the right to decide their destiny in the workplace," said Redman. "Nurses in many states across the nation have told us they are ready now to organize, and the ANA has allocated half a million dollars to state nurses associations to train organizers and help increase their organizing efforts."

"The ANA is uniquely positioned to handle an increased demand by nurses for assistance in determining workplace issues. We are the only national organization offering collective bargaining that is run by nurses for nurses," added Redman.

Redman said the ruling will lead to a stronger voice for nurses in promoting higher quality care for patients, adding value to the health care dollar.

Knowledge of Diagnosis and Reproductive History among Survivors of Childhood Plumbism

ABSTRACT

As part of a study of the long-term consequences of childhood plumbism, 35 survivors of lead poisoning in the period 1930 through 1944 were interviewed along with 22 controls matched for age, sex, and town of residence. Subjects were asked to recall all pregnancies they engendered (men) or carried (women), outcome, and intellectual development of resulting children. Matched female plumbism subjects reported a higher proportion of spontaneous abortion or stillbirths among pregnancies (relative risk = 1.60, 95% confidence interval [CI] = 0.6–4.0) and a higher proportion of learning disabilities among school-age children (relative risk = 3.0, 95% CI = 0.9–10.2) in comparison with their controls. There was no evidence of clustering of learning disabilities within families or confounding by parental educational level. These results suggest that women with a history of childhood lead poisoning may be at risk for having spontaneous abortions or stillbirths and having children who manifest significant learning disabilities. (*Am J Public Health.* 1991;81:1070–1072)

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Introduction

Lead accumulated from past exposures is not inert, as previously thought; rather, it is probably released continually from storage sites.[1,2] More than 95% of stored lead in an adult is located in the skeleton. Recent mammalian animal studies have suggested that a significant fraction of skeletal lead content is mobilized, enters the circulation, and crosses the placenta during both pregnancy and lactation.[3,4]

Since lead is a well-known behavioral teratogen, even at relatively low levels of exposure, and skeletal lead stores are known to persist for decades, the possibility exists that lead can be a delayed transgenerational toxin, i.e., it can affect the next generation even after maternal exposures have long ceased.

We report results of a reproductive questionnaire administered to subjects participating in an investigation of the long-term consequences of childhood plumbism (lead poisoning). We also report the level of awareness that survivors of childhood plumbism had with regard to personal histories of lead exposure. All research described herein was approved by the institutional review boards of the Brigham and Women's Hospital, Boston Children's Hospital and Medical Center, and the Harvard School of Public Health.

Methods

Methods used to define, trace, and recruit eligible subjects are described in detail elsewhere.[5] In summary, we used microfilmed medical records to identify 192 subjects admitted to Boston Children's Hospital during the period 1930 to 1944 who met the following criteria for lead poisoning: (1) a definite history of pica for lead-containing material (usually paint) or the use of lead nipple shields, (2) evidence of dense metaphyseal bands ("lead lines") on at least one long bone x-ray, (3) clinical evidence of symptoms and signs characteristic of childhood lead intoxication, and (4) the absence of clinical evidence for rickets. Subjects with continued residence in Massachusetts were traced using available public records.

The current status of 145 (76%) subjects was ascertained. Of these, 102 were found with current Massachusetts addresses. The 72 who lived in the Boston area were sent letters recruiting them for a "study on the impact of the environment on health."

Thirty-five subjects participated in the study. In general, participants had a history of less severe plumbism than nonparticipants.[5] Also enlisted were 22 control subjects matched for age, sex, and neighborhood. Town books were used to identify eligible controls, and letters of recruitment were sent. Budget constraints prevented recruitment of matched control subjects for 13 of the childhood plumbism subjects.

Each participant received a package containing a self-administered questionnaire and instructions. The questionnaire included detailed medical, occupational, and environmental history components. Each subject was asked to list all known pregnancies (or engendered pregnancies, in the case of males) with dates; whether the pregnancy resulted in a miscarriage, still birth, live birth, or was voluntarily aborted; and the sex of live births. For each offspring that survived to school age, the subject was asked whether he or she had had a learning disability. This was defined as difficulty in school that either (1) resulted in being left back one or more grades or (2) required special in-school remedial education. Learning disability de-

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This paper was submitted to the journal July 2, 1990, and accepted with revisions February 20, 1991.

fined similarly has been found to be associated with elevated dentine levels in a longitudinal study of teenagers.[6]

Results

Matched plumbism subjects did not differ appreciably from either the matched controls or the nonmatched plumbism subjects with respect to age, sex ratio, years of education, smoking status, alcohol ingestion, and medication use (Table 1).

In response to questions on childhood illnesses, 13 (37%) of all plumbism subjects noted being unaware of having had lead poisoning as children.

Among all plumbism subjects, women had 19 miscarriages or stillbirths out of a total of 79 pregnancy outcomes (24%), and 13 children with learning disabilities out of 60 school-age children (22%) (Table 2). Matched female plumbism subjects had 11 miscarriages or stillbirths out of a total of 51 pregnancies (22%), and 10 children with learning disabilities among a total of 40 school-age children (25%). Matched control subjects had 6 miscarriages or stillbirths out of 48 pregnancy outcomes (13%), and 3 learning-disabled children out of 42 school-age children (7%). The relative risk of matched female plumbism subjects for having a miscarriage or stillbirth was 1.60 (95% confidence interval [CI] = 0.6–4.0) and for having a learning disabled child, 3.0 (95% CI = 0.9–10.2). Inclusion of unmatched plumbism subjects did not appreciably alter these estimates. The pregnancies engendered by both the matched plumbism and control male subjects had few adverse reproductive outcomes. There were too few matched pair outcomes to offer meaningful analysis using McNemar's test.

TABLE 1—Participant Demographics and Severity Scores

Characteristic	Matched Plumbism Subjects	Matched Control Subjects	Unmatched Plumbism Subjects
n	22	22	13
Age			
Range (years)	49–60	48–60	47–60
Mean (years) ± SD	54.7 ± 4.0	55.4 ± 4.0	56.7 ± 4.0
Ratio of males to females	8/14	8/14	5/8
Education			
Range (years)	8–20	8–18	11–18
Mean (years) ± SD	13.5 ± 2.8	14.3 ± 2.9	12.9 ± 1.9
Smoking			
Never smokers	8	7	5
Former smokers	11	12	5
Current smokers	3	3	3
Usual alcohol ingestion			
<2 Alcoholic drinks per day	19	20	12
≥2 Alcoholic drinks per day	3	2	1

Discussion

The developing fetus and infant is clearly susceptible to the neurobehavioral effects of lead exposure. Efforts are currently under way to decrease such susceptibility by decreasing maternal exposures that have been associated with elevated lead levels in the blood, such as the use of leaded gasoline,[7,8] smoking and alcohol use,[9] and occupational exposures.[10]

Although this inquiry is of a small scale and produced results of borderline significance defined statistically, it provides evidence that remote exposures such as childhood plumbism may cause subsequent risk to a fetus or infant and result in later impairment of school performance. The relative risks found in this study may be underestimates, since study participants had been less severely intoxicated than nonparticipants.

This research also suggests that a significant fraction of individuals who had been diagnosed with lead toxicity as children are unaware of this history.

The presence of significant bias in this study seems unlikely. Other than a childhood history of plumbism, matched subjects did not differ from controls in terms of age, years of education, medication use, smoking, or alcohol ingestion. Studies that have examined the consistency of reproductive history questionnaires have generally not found significant recall bias.[11] Our analysis did not account for familial aggregation of adverse outcomes.[12] However, no more than two learning disabilities were observed in any one family in this study.

These results justify additional research efforts in this area. Overexposure to lead is still a widespread phenomenon among our society's children.[13] If lead poisoning poses a risk not only to these children, but also to these children's children, it may be important to screen young women entering reproductive age for lead burden and develop a strategy for prevent-

TABLE 2—Summary of Reponses to Reproductive Questionnaire

	Pregnancies No.	Miscarriages or Stillbirths No.	Percentage	Children Alive at School Age	Children with Learning Disabilities No.	Percentage
Females						
Matched plumbism subjects	51	11	22	40	10	25
Matched control subjects	48	6	13	42	3	7
Nonmatched plumbism subjects	28	8	29	20	3	15
Males						
Matched plumbism subjects	30	1	3	29	3	10
Matched control subjects	30	3	10	27	2	7
Nonmatched plumbism subjects	16	0	0	16	1	6

ing mobilization and/or transfer of accumulated lead to the fetus. History-taking may not be sufficient to identify women at risk. □

Acknowledgments

This research was supported by National Institutes for Environmental Health Sciences Center (NIEHS) Grant ES00002, NIEHS National Research Service Award 2T 32ES 07069, and National Institute of Health Clinical Research Center Grant RR 02635. The project would not have been possible without the diligent tracing efforts of Nancy Potter and her assistants at the Dana Farber Cancer Center. Many thanks go to Drs. Richard Monson, Stephen Lagakos, David Christiani, John Graef, and Fred Li for expert advice; Sarah Freeman, Kim Damakosh, and Cynthia Cryan for help in recruiting traced subjects; and Susan Fisher, RN, in evaluating subjects.

An abstract of this research was presented at the August 1990 meeting of the International Society for Environmental Epidemiology in Berkeley, CA.

References

1. Rabinowitz MB, Wetherill GW, Kopple JD. Kinetic analysis of lead metabolism in healthy humans. *J Clin Invest.* 1976; 58:260–270.
2. US Environmental Protection Agency. *Air Quality Criteria for Lead, I–IV.* Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office's; 1986. EPA Report No. EPA-600/8-83/028aF-dF. Available from National Technical Information Service, Springfield, Va. Report No. PB87-142378.
3. Keller CA, Doherty RA. Bone lead mobilization in lactating mice and lead transfer to suckling offspring. *Toxicol Appl Pharmacol.* 1980;55:220–228.
4. Keller CA, Doherty R. Lead and calcium distributions in blood, plasma, and milk of the lactating mouse. *J Lab Clin Med.* 1980;95:81–89.
5. Hu H. A fifty year follow-up of childhood plumbism: hypertension, renal function, and hemoglobin among survivors. *Am J Dis Child.* In press.
6. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood—an 11-year follow-up report. *N Engl J Med.* 1990;322:83–88.
7. Rabinowitz M, Needleman HL. Temporal trends in the lead concentration of umbilical cord blood. *Science.* 1987;216:1429–1431.
8. Rabinowitz M, Needleman HL. Petrol lead sales and umbilical cord blood lead levels in Boston, Massachusetts. *Lancet.* 1983;1:63–65.
9. Ernhart CB, Wolf AW, Sokol RJ, Brittenham GM, Erhard P. Fetal lead exposure: antenatal factors. *Environ Res.* 1985;38:54–66.
10. Wang JD, Shy WY, Chen JS, Yang KH, Hwang YH. Parental occupational lead exposure and lead concentration of newborn cord blood. *Am J Ind Med.* 1989;15:111–115.
11. Mackenzie SG, Lippman A. An investigation of report bias in a case-control study of pregnancy outcome. *Am J Epidemiol.* 1989;129:65–75.
12. Wilcox AJ, Gladen BC. Spontaneous abortion: the role of heterogeneous risk and selective fertility. *Early Hum Dev.* 1982;7:165–178.
13. Agency for Toxic Substances and Disease Registry. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Washington, DC: Public Health Service, US Department of Health and Human Services; July 1988.

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American Journal
of Public Health
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spect to the AIDS epidemic: (1) Could an immediately available, nonpunitive maintenance program attract into treatment a significant number of presently unmotivated addicts and reduce their risk behavior? (2) If a large number responded, could clinics of this kind expand fast enough to meet demand, while keeping a balance with other programs providing full service? The authors approached these questions by restricting the study group to addicts on the waiting list of an established full-service program, with the understanding that they would be released from the study when an opening became available in the conventional program.

Even with this reduction in scope, the study was vigorously opposed by the treatment community and by the New York State Division of Substance Abuse Services—a result that provided an unequivocally negative answer to the question of feasibility. Although in principle it might be argued that addicts are a heterogenous mixture of persons with widely different social needs and therefore that a range of different programs (including special facilities for pharmacological induction and social evaluation during the first month of treatment) are needed for the efficient use of resources, the treatment community and governmental administration have spoken with a virtually unanimous voice: Minimum-service programs will be prohibited, even as preliminaries to full-service programs.

On May 23, 1991, the US Public Health Service working group on methadone treatment rescinded plans for a regulatory change that would have permitted the operation of interim facilities for addicts on waiting lists of licensed clinics. The goal for the future, apparently, is to eliminate the embarassment of waiting lists by discouraging narcotic addicts from applying for treatment. ☐

Vincent P. Dole, MD

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Reference

1. Yancovitz SR, Des Jarlais DC, Peyser NP, Drew E, Friedmann P, Trigg HL, Robinson JW. A randomized trial of an interim methadone maintenance clinic. *Am J Public Health.* 1991;81:1185–1202.

Errata

In: Pass KA, Schedlbauer LM, MacCubbin PA, Glebatis DM. III. Comparison of newborn screening records and birth certificates to estimate bias in newborn HIV serosurveys. *Am J Public Health*. May 1991;81(suppl):22–24.

In table III-1, on page 23, the region labels are reversed. The first two lines of data in the left column should be labeled "NYS Exclusive of NYC"; and the third and fourth lines should be labeled "NYC."

* * *

In: Glebatis DM, Novick LF, Stacy A. VIII. Hospitalization of HIV-seropositive newborns with AIDS-related disease within the first year of life. *Am J Public Health.* May 1991;81(suppl):46–49.

Four lines were omitted at the bottom of column 2 on page 46. The complete paragraph should read: "A total of 995 HIV-seropositive newborns born in December 1987 through June 1988 were confirmed in the Newborn HIV Seroprevalence Study. A total of 151 infants born during this time period were hospitalized with an ARD within 12 months of birth. Assuming that all members of the hospitalized set were members of the seroprevalence set, 15 percent of HIV-seropositive infants born in these seven months were hospitalized with an ARD within 12 months of birth. The monthly ratios ranged from 14 to 18 percent (Table 1)."

Adult Foster Care for the Elderly in Oregon: A Mainstream Alternative to Nursing Homes?

Rosalie A. Kane, DSW, Robert L. Kane, MD, Laurel Hixon Illston, MSPH, John A. Nyman, PhD, Michael D. Finch, PhD

ABSTRACT

Background. **In Oregon, adult foster care (AFC) homes, which are private residences where a live-in manager cares for one to five disabled residents, have been covered by Medicaid since 1981 and seem to offer a mainstream alternative to nursing homes. They house almost 6000 older people, two thirds of which pay privately.**

Methods. **In a cross-sectional study, we interviewed 400 AFC and 400 nursing home residents. Data analyses included descriptive cross-tabulations; hierarchical loglinear models for judging the effects of care setting and payment status on resident characteristics; and logit analyses for predicting care setting and payment status within care settings.**

Results. **On average, nursing home residents were more physically and cognitively impaired than AFC residents, but there was considerable overlap in patterns of frailty in the two settings. Medicaid AFC residents were less disabled than privately paying AFC residents. AFC residents reported more social activity, even when we controlled for disability status. AFC residents and their families were more likely to value privacy and homelike settings when choosing a care setting, whereas nursing home residents were more likely to value rehabilitation and organized activity programs.**

Conclusions. **Both AFC and nursing homes are viable components of a long-term care repertoire. The greater disability levels of private-pay AFC residents refutes the criticisms that disabled Medicaid residents were being inappropriately channeled to AFC. (*Am J Public Health.* 1990;81:1113–1120)**

Introduction

The repertoire of suggested care to supplant or supplement nursing homes has been growing, although demonstration research thus far has not shown the cost-effectiveness of such care in major randomized trials.[1–3] Lackluster results of demonstration projects are attributed, in part, to poor targeting of the new community care programs to people unlikely to enter nursing homes or to stay there for long. It is also inherently difficult to deliver home care as efficiently as nursing home care to people with heavy care needs. Economies of scale are required, and this, in turn, requires residential proximity of those needing care, which is certainly a characteristic of nursing homes.

This line of reasoning has led to enhanced interest in developing residential settings for functionally impaired persons that permit economies of scale in their care but allow the older disabled persons to preserve personal autonomy and privacy and have more control over their routines. In such living settings, the resident is not viewed first and foremost as a patient.

Study Setting

Adult foster homes, as they have developed in Oregon, provide one such residential setting. An adult foster home in Oregon is a private residence in a residentially zoned area that is licensed to care for one to five disabled adults. Each foster home must have a live-in resident manager (who may also own the home). Meals are served family style, and regulations are kept to a minimum to enhance a homelike atmosphere and autonomy for the residents. The resident manager provides personal care and housekeeping services. If necessary, other services, such as skilled nursing, can be brought into the foster home from home health agencies.

Although the idea of adult foster care is inherently attractive as a concept, several demonstrations projects in other states have failed to develop a strong market for the service.[4–8] However, Oregon's experience with adult foster care has been different in two ways. First, although Medicaid clients were the first to widely use adult foster care, by 1988 private-paying customers constituted more than two thirds of the nearly 6000 residents in foster care. Second, and related, in other states where foster care exists, the programs tend to be proportionately much smaller and largely serve low-income clients with mental illness or developmental disabilities.[9] Only in Oregon does adult foster care seem a mainstream possibility for the elderly.

The growth of adult foster homes in Oregon was stimulated in 1982 when the state made adult foster care a benefit under its Medicaid waiver program. To receive subsidies under this program, the adult foster care resident needed to be eligible for nursing home care on the basis of functional disabilities. The state developed a vigorous program to divert Med-

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This paper was submitted to the journal December 10, 1990, and accepted with revisions May 1, 1991.

icaid clients already in nursing homes to foster homes when the routes to home had been cut off and urged clients who could no longer remain at home to consider adult foster care as an alternative to nursing home care. By 1988, adult foster care was widely accepted by the public as a desirable means of caring for older persons in need of a supportive environment.

Several facts about adult foster care in Oregon were clear. First, the growing utilization suggested that many elderly and families found adult foster care a desirable option. Second, the success of adult foster care within both the private and Medicaid markets (along with vigorous home care programs) was sufficient to reduce the demand for nursing home beds. Between 1981 and 1989, the supply of nursing home beds per 1000 persons aged 75 years and older fell from 50 to 40, and the occupancy rate decreased from 92% to 87%.[10] Oregon is the only state where nursing home use dropped in absolute as well as proportional terms. Third, adult foster care is a less expensive program than nursing home care; both the Medicaid rates and the average private pay rates in foster care were about 2/3 of those in nursing homes. And fourth, adult foster homes became the focal point for criticism from the nursing home industry, which claimed that the care was inadequate, that the degree of social stimulation was insufficient, and that the Medicaid program, in particular, was denying recipients access to the appropriate level of care.

In this context, the state of Oregon commissioned the University of Minnesota to evaluate the adult foster care program. The study had several components, including a mailed census of the foster care industry and a retrospective study of change over time using program data for the Medicaid population. Both of these will be reported in future papers. This paper reports the results of a cross-sectional comparison of 400 foster care residents and 400 nursing home residents. Our analysis looked at differences between care settings and differences between Medicaid and private pay clients within care settings. The ramifications of these questions for public policy extend beyond Oregon at a time when serious consideration is being given to tightening the regulation of group homes and some authorities argue that elderly people should be channeled to particular care settings on the basis of measurable functional characteristics.

Method

Sample

Between December 1988 and February 1989, we conducted in-person, structured interviews with 402 intermediate care facility (ICF) residents and 405 foster home residents in four geographic areas, the latter chosen to represent different facets of long-term care delivery in Oregon (i.e., rural vs urban, Medicaid administration by area agencies on aging vs by regional state offices of the Senior Services Division) while reflecting the average, typical state experience on descriptors such as proportion of elderly, and adult foster home and ICF bed-to-population ratios. Original data collection was necessary because, although considerable data are available about Medicaid residents living in adult foster homes and in nursing homes through their periodic assessments by case managers, little existing data are available on the private pay group in either setting, particularly in foster homes. Moreover, even the Medicaid data set has no information on social activities or choice of care setting.

We generated the foster care sample by randomly selecting foster homes in each geographic area and enrolling all residents of the selected foster homes until we reached the desired proportional number of respondents for each of the four regions. Enough nursing homes were selected randomly in each geographic area to allow us to interview 10 residents in each facility and achieve a number of subjects equal to the foster home sample. From a list of all ICF residents in the randomly selected homes, we randomly selected 5 Medicaid and 5 private pay ICF residents, thus reflecting the proportion of Medicaid and private pay residents in the population. (The nursing home sample was limited to ICF residents because the skilled nursing care level in Oregon is a very small program and widely agreed to include only very disabled and medically unstable persons who are not appropriately compared to adult foster home residents).

To reach the desired sample size of at least 400 residents in each care setting, 108 foster homes were included in the sample; only 4 foster homes approached refused to participate. The final nursing home sample included 39 facilities; only 2 nursing homes approached refused to participate. Participation of the sampled residents was also very high. Actual refusals to participate represented only 4%—6 ICF residents and 5 family members on behalf of a cognitively impaired ICF resident declined to participate. No refusals occurred among foster care residents.

Data Collection

Interviewers were recruited from the local areas and trained by University of Minnesota staff. Most had previous experience in human services or health care. Background information such as date of admission, payment status, medical diagnosis, and family member most knowledgeable about the resident (should a family proxy be needed) was first gathered from the care setting staff on each sample member. For this purpose, records were consulted when necessary. If the resident in either care setting seemed to be an unreliable informant, the interviewers were instructed to skip to a later section of the schedule and attempt to administer the mental status questionnaire. When few of the 10 MSQ questions were answered correctly, or when the resident was clearly too confused to answer, the interviewers moved to proxy respondents. Questions about choice of care setting and satisfaction with care setting were administered by telephone to a family member. The foster care home's resident manager or a nursing home staff member who knew the resident best served as the proxy respondent for questions about current physical functioning, activities, and recent use of hospitals, emergency rooms, and physicians.

Instruments

Identical questions were asked of adult foster home and ICF residents (merely interchanging the terms "adult foster home" and "nursing home" when appropriate.) The functional abilities of the residents were measured through questions carefully designed to be compatible with those found in the SSD 360 (the comprehensive assessment format used by case managers to provide a database on all Medicaid waiver clients in Oregon.) Activities of daily living and instrumental activities of daily living (ADL/IADL) items included toileting, dressing, transferring, bathing, eating, ambulation, and internal medicine use. Cognitive status was measured by the short, portable mental status questionnaire. The health section included self-reported health, vision, hearing, self-diagnosis, and health care utilization. For this study we added three questions to tap mood (depression, anxiety, and positive affect) and questions on activities, recent relationships, satisfaction items, and items on factors related to choice of care setting adapted from an earlier study of nursing home outcomes.[11] The questionnaire and the Oregon SSD 360 are available from the authors. The instrument was characterized by its use of direct questions to respondents rather than by interviewer ratings of the attributes of the respondents.

Statistical Analysis

All data analyzed were categorical. Crosstabs were run on all data with hierarchical loglinear three-way chi-squares used to judge whether the main effects of client setting or payment status or their interactions were significant. Logit analysis was performed to determine which factors predict care setting and payment status within care setting.

Findings

Case-Mix Differences

As shown in Table 1, the demographic characteristics of the samples were similar across care settings, with payment status in foster care reflecting actual proportions in the population. We have separated the private and Medicaid supported clients in all analyses because of policy interest. There were, however, some substantial differences between the two groups. Foster care residents were less likely to come from acute care hospitals. Only 12% and 13% of Medicaid and private pay residents were admitted from a hospital, compared with 44% and 33% of corresponding nursing home patients. Fourteen percent of Medicaid and 17% of private adult foster care residents came from nursing homes, whereas only 9% of Medicaid residents and 7% of private nursing home residents came from adult foster homes.

Table 2 contrasts the pattern of diagnoses. The residents in nursing homes had more major health problems than those in adult foster care; yet this information must be interpreted with some degree of caution because some adult foster care providers had imprecise knowledge of their clients' diagnoses, and the records in adult foster care homes were less complete than in nursing homes. There were also differences by payment status. These did not follow as clear a pattern. For example, whereas clients with dementia were more frequently private pay in adult foster care, they were about equally distributed in nursing homes at a rate still higher. By contrast, patients with seizures were more

TABLE 1—Demographic Background of Sample

Characteristic	No. (%) of Adult Foster Home Residents (n = 405)	No. (%) of Nursing Home Residents (n = 402)
Gender		
Percent Female	314 (78)	293 (74)
Age, y		
Under 50	16 (4)	10 (2)
50–64	21 (5)	12 (3)
65–74	51 (13)	46 (11)
75–84	117 (29)	116 (29)
85–94	156 (38)	164 (41)
95+	45 (11)	54 (13)
Marital Status		
Married	39 (10)	78 (20)
Widowed	260 (67)	249 (64)
Divorced	53 (14)	24 (6)
Single	38 (10)	41 (11)
Education		
Grade school or less	169 (47)	165 (45)
Some or all high school	120 (33)	106 (29)
Some college	50 (14)	57 (16)
College or more	23 (6)	37 (10)
Payment Status		
Private	255 (63)	214 (53)[a]
Medicaid	150 (37)	188 (47)

[a] Although the original intent was to have equal distribution between Medicaid and private pay individuals in nursing homes, two homes drawn in the sample accept only private-pay residents.

TABLE 2—Major Health Problems

	Adult Foster Home		Nursing Home	
Medical Problem	Medicaid, % (n = 150)	Private, % (n = 255)	Medicaid, % (n = 188)	Private, % (n = 214)
Dementia	23	36	42	44
Heart disease	20	22	32	28
Arthritis	13	11	14	10
Stroke	16	23	21	21
Blood pressure	11	5	10	9
Skeletal/muscular	10	10	15	9
Mental illness	14	11	6	7
Vision	8	7	5	4
Hip fracture	3	5	7	17
Respiratory	9	4	10	5
Parkinson's	4	5	5	10
Diabetes	11	8	10	7
Alcoholism/drug abuse	7	1	3	3
Incontinence	4	3	2	3
Mental retardation/developmental disability	5	1	3	—
Old age	3	5	1	—
Hearing	3	5	2	2
Other neurological	3	1	5	5
Cancer	4	2	1	4
Seizures	3	2	9	2
Other bowel and bladder problem	1	1	8	11
Multiple sclerosis	1	1	2	3
Other diagnoses[a]	4	3	12	14
Other nondiagnostic problem[b]	10	7	3	5
Unknown to respondent	4	3	—	—

[a] This category includes any specific diagnosis that was mentioned less than 1% of the time.

[b] This category includes any "nondiagnostic" problem mentioned less than 1% of the time—e.g., weight loss.

TABLE 3—Functional Abilities by Care Setting and Payment Status

	Ability to Function, %											
	Without help				Some Help				Complete Help			
	Foster Home		Nursing Home		Foster Home		Nursing Home		Foster Home		Nursing Home	
ADL Function	Medicaid	Private	Medicaid	Private	Medicaid	Private	Medicaid	Private	Medicaid	Private	Medicaid	Private
Moving	56	48	27	22	34	29	22	25	9	23	52	53
Transferring	86	63	27	24	7	18	20	19	7	19	54	57
Bathing	33	17	6	7	34	32	19	17	33	51	75	76
Dressing	65	48	19	19	22	25	25	20	13	27	56	61
Eating	88	80	60	53	8	13	24	28	4	7	16	20
Toileting	81	65	26	23	12	16	19	17	7	19	54	60

ADL = activities of daily living.

TABLE 4—Cognitive Status by Care Setting and Payment Status

	Adult Foster Home, No. (%)				Nursing Home (ICF), No. (%)			
	Medicaid		Private		Medicaid		Private	
Five or more errors on the SPMSQ	38	(27)	44	(3)	42	(36)	52	(43)
Unable to administer the SPMSQ	11	(7)	58	(23)	67	(36)	87	(41)
Total	49	(34)	122	(56)	109	(72)	139	(84)

SPMSQ = Short Portable Mental Status Questionnaire.

likely to be Medicaid clients in nursing homes and developmentally disabled clients were more likely to be supported by Medicaid in both settings.

The extent of dependency in ADLs is shown in Table 3. More adult foster care clients were able to perform each of the six ADLs studied without assistance. Private paying residents in adult foster care were more disabled than those supported by Medicaid, whereas the proportions were more similar within the nursing home sample. This pattern of difference by payment status is also seen in the levels of cognitive performance on the 10-item Short Portable Mental Status Questionnaire (SPMSQ[12]; see Table 4). Among adult foster care residents, 33% of the private-pay clients had five or more errors, and another 23% could not respond to the test at all. Among the Medicaid foster home group, 27% had five or more errors, and only 7% could not respond. In the nursing home sample, 43% of the private-pay clients made five or more errors, and 41% could not respond; for Medicaid residents the comparable figures were 36% and 36%. Thus a total of 56% of private-pay adult foster care residents and 34% of Medicaid residents did poorly on the SPMSQ compared with 84% of private pay nursing home residents and 72% of Medicaid residents.

TABLE 5—Proportion Considering Selected Factors Important in Selection of Care Setting

	Adult Foster Home, %		Nursing Home, %	
Factor[a]	Medicaid (n = 150)	Private (n = 255)	Medicaid (n = 188)	Private (n = 214)
Homelike atmosphere	94	96	63	62
Safe, supervised place to live	93	96	94	92
Personal assistance	82	84	87	88
Privacy	80	82	54	54
Medical care	80	60	82	80
Flexible routines and rules	81	78	57	51
Cost	70	77	68	66
Location/neighborhood	68	67	69	72
Organized activities	33	26	52	38
Physical rehabilitation program	26	21	51	39
Compatibility or familiarity with facility, staff, or residents	9	4	1	5
Quality care and staff	4	5	8	10
Clean and pleasant surroundings	2	3	8	7
Other	6	10	5	5
None of the above	—	—	2	1

[a] Respondents could select more than one factor as important.

Other reflections of the differences across these settings can be seen in the amount and location of physician care (not shown in a table). Although the proportion of clients reporting a physician visit in the previous 6 months was similar by payment status, nursing home patients were more likely to see a physician. Among adult foster care residents, 80% of Medicaid and 77% of private residents saw a doctor in the previous 6 months compared with 90% of Medicaid and 91% of private-pay nursing home patients. Caution should be used in interpreting these figures because physician contact is mandated on a periodic basis for Medicaid nursing home clients. The location of these visits was quite different. Whereas about 90% of the adult foster care residents saw a physician in his or her office,

TABLE 6—Activities in the Care Setting in the Last Few Months by Functional Status, %

	Never				Sometimes				Often			
	Hi[a]		Lo[b]		Hi		Lo		Hi		Lo	
Activity	AFC	NH	AFC	NH	AFC	NH	AFC	NH	AFC	NH	AFC	NH
Watch television	8	17	35	42	27	39	35	31	65	44	30	28
Listen to radio, to music	43	35	45	28	32	34	30	39	25	32	25	33
Talk to other residents	6	7	39	38	35	47	30	38	59	47	32	24
Read books or newspapers	29	42	69	78	26	24	15	11	45	34	17	11
Engage in hobbies	66	82	94	93	17	12	6	5	17	6	—	2
Chat with staff	3	8	15	25	29	47	35	43	68	45	50	32
Play cards or games	80	57	94	90	14	28	4	8	6	16	2	2
Do activities organized by home	61	28	85	37	26	51	11	44	13	21	4	19
Help with cooking/ housekeeping	68	94	100	98	16	2	—	1	17	3	—	1

AFC = adult foster care; NH = nursing home.
[a] Residents who did not need complete help on more than bathing (n = 290 in foster homes and n = 126 in nursing homes).
[b] Residents who need complete help with four or more functional activities (n = 54 in foster homes and n = 211 in nursing homes).

TABLE 7—Frequency with Which Residents Engaged in Activities Outside the Home, Adjusted for Functional Status in Foster Home and Nursing Home, %

	Hi[a] Status		Low[b] Status	
	AFC	NH	AFC	NH
Frequency				
Not at all	18	48	72	83
Less than once a month	30	26	20	13
Less than once a week	13	11	—	2
About weekly	26	11	7	1
Almost every day	13	5	—	—
Activity				
Take a walk in the neighborhood	36	34	—	11
Use a bus or public transportation	12	17	—	3
Go for a ride in a car	95	92	87	91
Visit relatives or friends	78	80	80	71
Attend church or other religious activities	29	18	—	24
Attend a senior center or club	17	9	7	—
Go shopping	52	34	20	14
Go to a restaurant, movie, or other entertainment	69	59	47	38
Attend activities outside the home arranged by the foster home	31	32	13	23

AFC = adult foster care; NH = nursing home.
[a] Residents who did not need complete help on more than bathing (n = 290 in foster homes and 126 in nursing homes).
[b] Residents who need complete help with four or more functional activities (n = 54 in foster homes and 211 in nursing homes).

only about 30% of the nursing home patients did so.

Choice of Care Setting

Mostly, the preferences of those living in adult foster care were similar to those of nursing home residents. As shown in Table 5, however, there were some important differences. For example, adult foster home residents were more concerned about a homelike atmosphere and privacy, whereas the nursing home residents emphasized organized activities and physical rehabilitation. Not surprisingly, both groups valued safety and personal assistance. In choosing one setting over another, 60% of the foster care residents and 36% of the nursing home residents (or their family proxies) believed that they had at least some control over the choice of care setting. A nursing home had been considered by 20% of foster care residents and a foster home had been considered by 15% of nursing home residents.

Living Conditions

The differences in the two settings can also be seen in the way in which residents spent their time. Table 6 describes the residents' activities according to their level of functioning in their respective care settings. Better functioning adult foster care residents were more likely to watch television, talk with other residents, and help with cooking and housekeeping, whereas better functioning nursing home residents were more likely to participate in organized activities and games and listen to the radio. As shown in Table 7, adult foster care residents were more likely to leave the care setting for activities other than medical appointments, especially shopping and entertainment, a finding that held true when we controlled for functional status and for cognitive status. The reversal of the usual pattern for low functioning nursing home patients reflects the fact that nursing home staff is more likely to organize group activities or to bring activities to the residents. The patterns of responses in Table 8 suggest that adult foster care residents were less likely to get visitors than nursing home residents but more likely to have telephone contact. Nearly 25% of the nursing home residents compared with only about 10% of adult foster care residents had a visitor every day. Only about 5% to 10% of both groups had no visitors. Telephone access was more difficult in nursing homes. About 33% of adult foster care residents com-

TABLE 8—Frequency of Visitors and Phone Contacts in the Last 6 Months by Cognitive and Functional Status in Foster Homes and Nursing Homes, %

	Cognitive Status				Functional Status			
	Hi[a]		Lo[b]		Hi[c]		Lo[d]	
	AFC	NH	AFC	NH	AFC	NH	AFC	NH
Have visitors								
Not at all	6	0	5	9	6	8	6	10
Less than once a month	26	13	24	25	28	16	17	23
Less than once a week	18	17	20	17	18	20	15	16
About weekly	41	32	39	31	39	34	49	29
Almost every day	9	29	12	18	9	22	13	22
Telephone contact								
Not at all	20	50	54	85	26	49	67	83
Less than once a month	22	15	22	7	25	13	9	6
Less than once a week	10	11	9	2	10	12	6	1
About weekly	30	13	10	5	25	15	9	4
Almost every day	17	12	4	2	14	11	9	3

AFC = adult foster care; NH = nursing home.

[a] Residents who answered four or fewer questions on the Short Portable Mental Status Questionnaire (SPMSQ) incorrectly (n = 235 in foster homes and 155 in nursing homes).

[b] Residents who answered five or more questions on the SPMSQ incorrectly or were unable to answer any SPMSQ questions (n = 169 in foster homes and 249 in nursing homes).

[c] Residents who did not need complete help on more than bathing (n = 290 in foster homes and n = 126 in nursing homes).

[d] Residents who needed complete help with four or more functional activities (n = 54 in foster homes and n = 211 in nursing homes).

TABLE 9—Satisfaction among the Cognitively Intact[a] in Foster Homes and Nursing Homes, %

	Very Satisfied		Satisfied		Unsatisfied		Very Unsatisfied	
	AFC	NH	AFC	NH	AFC	NH	AFC	NH
The food	47	23	47	54	4	18	1	5
Your room	46	31	50	61	3	7	1	1
Your roommate	30	23	62	64	8	9	—	4
Your daily care	45	27	52	64	3	9	—	1
Your daily activities	26	21	65	69	8	9	—	1
The amount of time you see family/friends	33	23	49	57	14	15	4	5
Your medical care	39	27	57	63	2	8	3	1
Your physical safety	45	32	53	64	1	3	1	1
The safety of your personal possessions	44	21	53	58	31	16	—	6

AFC = adult foster care; NH = nursing home.

[a] Residents who answered four or fewer questions on the SPMSQ incorrectly (n = 229 in foster homes and 143 in nursing homes).

pared with about 70% of nursing home residents had no telephone contact. Both cognitive and functional status was related to telephone access in both settings, but were less discriminating for visitors.

Satisfaction was generally high in both groups. Table 9 presents data on the reported satisfaction ratings for a number of items among the cognitively intact respondents. In general, the adult foster care respondents expressed stronger levels of satisfaction and less dissatisfaction. The exception to this pattern was in the area of safety of personal possessions where the adult foster care respondents showed greater degrees of dissatisfaction despite a larger proportion also being very satisfied.

Foster care residents and nursing home residents were similar in their use of hospitals in the past 6 months (18% for foster care compared with 15% for nursing home care) though the reported stays for foster care residents were, on average, 1.5 days longer (7.2 compared with 5.7). Foster care residents used emergency rooms more than did nursing home residents (20% compared with 8%).

Predicting Care Setting and Payment Status

We performed discriminant analysis on selected variables to identify which distinguished between adult foster care residents and nursing home residents and to predict payment status within each of the care settings. We have characterized the underlying care needs of our population with selected ADL and IADL variables: mobility, transferring, bathing, dressing, feeding, toileting, a summary score, and taking medicines. It was also hypothesized that a number of diagnoses would play a role in distinguishing between types of residents (residents with heart disease, skeletal-muscular problems, stroke, hip fracture, cancer, diabetes, incontinence, vision problems, hearing problems, and mental illness). To capture cognitive functioning, we included a variable for whether a proxy was used for the interview. We have also included five variables that describe where the client was living or from where he or she was admitted prior to coming to the setting: in a relative's home, in an adult foster home, in a residential care facility, in a nursing home, or from the hospital. Finally, we included two other demographic variables in the analysis: marital status and gender.

Table 10 shows that, of the functioning status variables, transferring, toileting, and taking medicines were significant in explaining the likelihood of a client's entering an adult foster home, whereas none predicted nursing home residency. Women were almost 19% more likely to be in adult foster homes. Residents who had previously lived in their family's home or other foster homes were 15% to 35% more likely to be in adult foster care. Only hip fractures and being on publicly subsidized care explained the likelihood of entering a nursing home.

Two variables, transferring and bathing, were important in estimating that an adult foster care resident is receiving Medicaid (see Table 11). In fact, residents needing help with transferring were more than 38% more likely to be in foster homes than in nursing homes. Marital status alone helped estimate clients with private sources of payment.

Table 12 shows that in a nursing home Medicaid payment status can be predicted by the presence of skeletal-muscular disease and the ability to take medicines. Privately paying residents, on the other hand, are more likely to be married, to have had cancer or a hip fracture as a diagnosis, and be incontinent.

By using hierarchical loglinear models to explain differences between care setting or payment status, we determined that none of our main effects or first level interactions was found to be significant.

TABLE 10—Variables That Explain the Likelihood of a Person's Entering an Adult Foster Home[a]

	b	Significance	Percentage[b]
Being female	.79778	<.01	17.1
Having previously lived in relative's home	1.11529	.03	27.1
Having previously lived in adult foster home	1.56264	<.01	37.2
Being Medicaid recipient	−.89297	<.01	−19.9
Being able to transfer	1.18578	<.01	27.3
Being able to toilet	1.32605	<.01	30.5
Being able to take medicines	.674659	.03	16.2
Having hip fracture	−1.22585	<.01	−23.1

[a] The logit analysis was weighted to reflect the actual distribution of adult foster home care in the population, and appropriate standard errors were compiled following Manski and McFadden (1981).[13]

[b] This represents the percent change in the probability of adult foster home placement as opposed to nursing home placement for people having the specified characteristics.

TABLE 11—Variables That Explain the Likelihood of a Person's Being a Medicaid Client in an Adult Foster Home

	b	Significance	Percentage[a]
Having previously lived in nursing home	1.0741	.02	26.1
Being able to transfer	1.6591	<.01	33.6
Being able to bathe	.9386	.01	22.7
Having questionnaire completed by proxy	1.0877	.03	22.3
Being married	−1.1066	.03	−22.0

[a] This represents the percent change in the probability of an adult foster home resident's being supported by Medicaid rather than by private means when these residents have the specified characteristics.

Discussion

These data corroborate impressions that, although adult foster care serves many elderly people with disabilities, the two forms of care serve rather different groups of the frail elderly. The patterns of frailty overlap to a considerable degree, but adult foster care treats a population that is, on the average, less physically and cognitively dependent than that found in nursing homes. Part of this effect may be due to the displacement that has already occurred, but at least part of it corresponds to the care needs of two distinct groups, at least at the ends of the distributions. Private-pay residents in foster care, however, show more functional impairment and cognitive impairment than do the Medicaid residents, making implausible the contention that state policy was driving Medicaid residents to accept a lower level of care than they might choose. Because the study is cross-sectional, we cannot say whether this discrepancy is due to more impairment among private-paying clients at admission or to a lesser likelihood that the residents are discharged to a nursing home, or to both.

It seems unlikely that adult foster care will replace nursing home care completely, and there will continue to be a need for both services. But adult foster care will probably continue to serve some (and perhaps a growing number) of persons who could also be legitimately served in nursing homes. Categorizing client needs in long-term care remains an imprecise activity, permitting a great deal of discretion in care planning. The policy issue becomes one of whether long-term care will be envisaged as a continuum with a niche for each client or whether it will be viewed as a repertoire of services that allows considerable choice based on lifestyle preferences and prices. A related policy issue cast in relief by this study is whether socially oriented care settings such as adult foster care in Oregon should be more heavily regulated to protect the residents at the risk perhaps of transforming the settings from homes to mini-nursing homes. This situation is exacerbated by the phenomenon of aging in place. Although the admission cohorts to the two forms of care may look more dissimilar, with time they grow increasingly to resemble each other and lead one to conclude that the care is interchangeable. It would, thus, appear both impractical and undesirable to formulate policies based on a rigid delineation of the "appropriateness" of the particular resident for the particular setting. It appears from our data that residents and their families at least in part shop for the setting that is "appropriate."

Moreover, adult foster care has proven attractive to the private market. It is important to appreciate that the growth of this sector in Oregon has not been the result of pressure from the Medicaid agency. Clients and their families able to choose the location of their care have opted for the greater privacy and ambiance and lower price of the adult foster home.

The apparent success of adult foster care has spawned concerns about the need for stricter regulation of this industry on a level more akin to that applied to nursing homes. This pressure is understandable, but it threatens the essence of the program; that is, an approach that relies on an extended family type of arrangement for most care and community agencies for the more technically sophisticated services.

From this cross-sectional study, it was impossible to examine change over time for residents in either care setting, although it appeared that, even when we controlled for disability, foster care residents enjoyed somewhat more social and community activity. In a forthcoming paper, we will compare change in functional outcomes for Medicaid foster care residents and nursing home residents, using the secondary data base available in the Medicaid program. The results of the present analyses offer support to those who are enthusiastic about the adult foster home as a viable option for some subset of those ordinarily served in nursing homes, while recognizing that on average the nursing home residents are more disabled. Indeed, the presence of the foster care program (and other home care programs not discussed in this paper) makes it likely that the nursing home population will be

TABLE 12—Variables That Explain the Likelihood of a Person's Being a Medicaid Client in a Nursing Home

	b	Significance	Percentage[a]
Having a skeletal muscular problem as diagnosis	.9363	.02	22.9
Having a hip fracture as diagnosis	−1.2999	<.01	−28.2
Having cancer as diagnosis	−2.1125	.02	−36.9
Having incontinence as diagnosis	−.7965	.02	−19.0
Being able to take medicines	1.0284	.05	24.9
Being married	−.6693	.04	−15.9

[a] This represents the percent change in the probability of a nursing home resident's being supported by Medicaid rather than by private means when the residents have the specified characteristics.

more disabled than nursing home populations in other states. ☐

Acknowledgments

This study was supported by a grant from the Oregon Senior Services Division, and additional assistance was provided by the Administration on Aging and the Southmark Foundation.

An earlier version of this paper was presented at the 42nd Annual Meeting of the Gerontological Society of America, Minneapolis, Minn, November 1989.

References

1. Kane RA, Kane RL. *Long-Term Care Principles, Programs and Policies.* New York, NY: Springer Publishing Company; 1987.
2. Thornton C, Dunstan SM, Kemper P. The evaluation of the national long-term care demonstration: the effect of channelling on health and long-term care costs. *Health Serv Res.* 1988;23:129–142.
3. Weissert WG, Cready CM, Pawelak JE. The past and future of home- and community-based long-term care. *Milbank Q.* 1988;66:309–388.
4. Braun K, Rose C. The Hawaii geriatric foster care experiment: impact evaluation and cost analysis. *Gerontologist.* 1986;26:516–523.
5. Mor V, Sherwood S, Gutkin C. A national study of residential care for the aged. *Gerontologist.* 1986;26:405–417.
6. Oktay JS, Volland PJ. Foster home care for the frail elderly as an alternative to nursing home care: an experimental evaluation. *Am J Public Health.* 1987;77:1505–1510.
7. Sherman SR, Newman ES. *Foster Families for Adults: A Community Alternative in Long-Term Care.* New York, NY: Columbia University Press; 1988.
8. Sherwood S, Morris JN. *Pennsylvania Domiciliary Care Pilot Program.* Hebrew Rehabilitation Center for Aging, 1983.
9. Kane RA, Illston LH, Kane RL, Nyman JN. *Meshing Services with Housing: Lessons from Adult Foster Care and Assisted Living in Oregon.* John A. Hartford Foundation, 1990.
10. Saslow MG, Dietsche S. *Oregon's Public Long Term Care System.* Oregon Department of Human Resources, Senior Services Division; 1988.
11. Kane RL, Bell R, Hosek S, Riegler S, Kane RA. *Outcome-Based Reimbursement for Nursing Home Care.* The RAND Corporation; 1984.
12. Pfeiffer E: A Short Portable Mental Status Questionnaire for the Assessment of Organic Brain Deficit in Elderly Patients. *J Am Geriatr Soc.* 1975;23:433–411.
13. Manski C, and McFadden D. (eds). *Structural Analysis of Discrete Data with Econometric Application.* MIT Press; 1981.

International Workshop of Computing in Occupational and Environmental Health to Be Held in Paris in November 1991

The 3rd International Workshop of Computing in Occupational and Environmental Health will be held November 7 & 8, 1991, in Paris, France, at the Hotel Meridien-Porte Maillot.

Professionals in occupational and environmental health as well as computer scientists will compare their latest applications in the following areas:

- individual medical date records and process
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Is Coronary-Care-Unit Admission Restricted for Elderly Patients? A Multicenter Study

Craig Fleming, MD, Ralph B. D'Agostino, PhD, and Harry P. Selker, MD, MSPH

ABSTRACT

Background. To investigate whether elderly patients are more likely to experience restricted access to high technology medical care, we examined the impact of age on the likelihood of coronary care unit (CCU) admission for patients with acute myocardial infarction.

Methods. As part of a prospective investigation of emergency room triage for patients with suspected cardiac ischemia, we studied 4223 patients presenting to six hospitals. Because CCU admission is the accepted standard of care for acute infarction, we defined nonadmission to the CCU as a restriction of access to care. We used a logistic regression model to control for gender, hospital, and CCU occupancy at the time of admission and examined the relationship between age and CCU nonadmission.

Results. Patients 75 years or older with acute myocardial infarction were 2.5 times more likely not to be admitted to the CCU than younger patients (RR 2.5, 95% CI 1.64, 3.85). Coronary unit admission was restricted even when the physician's admitting diagnosis was "myocardial infarction" (RR 7.1, 95% CI 2.1, 25.0) or "rule-out myocardial infarction" (RR 1.5, 95% CI 1.1, 2.1). Observed differences in clinical presentation or severity of illness between older and younger patients did not account for these findings.

Conclusion. Our findings suggest that physicians may intentionally restrict access to coronary care for elderly patients with acute myocardial infarction. (*Am J Public Health.* 1991;81:1121–1126)

Introduction

As health care expenditures continue to grow, concerns about the cost of medical care for the aged have arisen.[1,2] People over 75 years of age constitute the most rapidly growing segment of our population.[3] Because per capita costs of health care for the elderly exceed those of younger people, further increases in health care expenditures seem inevitable as our population ages. Intensive-care-unit (ICU) care, which accounts for a substantial portion of hospital-related outlays, is considered a prime target for limiting health care expenditures.[4]

In response to these concerns, society and the medical profession have begun to question the appropriateness of unrestricted use of high technology medical care in the elderly.[5–7] While there is presently no consensus regarding appropriate limitations of such care, it has been suggested that the threshold to admit patients to ICUs may already be age-related, resulting in less frequent admission of older patients despite more severe disease among the elderly.[8] To examine this issue, we studied the impact of age on physicians' coronary care unit (CCU) admitting practices for emergency room (ER) patients with symptoms suggesting acute cardiac ischemia. Our objective was to determine if differences existed in CCU admission practices for older patients, as compared with younger patients, that could not be attributed to differences in clinical presentation or severity of illness.

Methods

The data on which this study was based were collected prospectively during a multicenter clinical trial to study CCU admitting practices and to develop and test an instrument predicting the likelihood of acute ischemic heart disease among patients in the ER setting. More detailed information regarding the data collection and design of the study may be found in the paper describing that study.[9]

Participating Hospitals

The six participating hospitals in the study included two urban medical-school teaching centers, two medical school-affiliated teaching hospitals in smaller cities, and two rural nonteaching hospitals.

Study Subjects

The study included all consenting patients, 40 years of age and older, presenting to the ERs of any of the six hospitals, with a chief complaint of chest, jaw, or left arm pain, shortness of breath, or other symptoms suggestive of acute ischemic heart disease. These criteria were based on the Imminent Myocardial Infarction Rotterdam Study and identify more than 90 percent of patients with acute cardiac ischemia who seek medical care.[10] Patients were asked to participate in a study of the diagnosis and treatment of heart disease, and among patients meeting the entry criteria, 92 percent consented to par-

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TABLE 1—Physicians' Triage Disposition for Younger and Older Emergency Room Patients with Acute Myocardial Infarction, Angina Pectoris, and without Acute Ischemic Heart Disease (N = 4,223)

	Triage Disposition						
	Admitted to Coronary Care Unit		Admitted to Ward		Discharged Home		Total
Final Diagnosis	n	%	n	%	n	%	n
Myocardial Infarction							
Age 40–74	557	89.8	45	7.3	18	2.9	620
Age ≥ 75	146	76.8*	42	22.1	2	1.1	190
Angina Pectoris							
Age 40–74	377	66.6	137	24.2	52	9.2	566
Age ≥ 75	82	57.0	51	35.4	11	7.6	144
No Acute Ischemic Disease							
Age 40–74	566	27.8	605	29.7	868	42.5	2,039
Age ≥ 75	183	27.5	373	56.2	108	16.3	664

*Relative risk (RR) of not being admitted to the CCU for older patients compared with younger patients is 2.50 (95% CI = 1.64, 3.85)

TABLE 2—Physicians' Triage Disposition for Patients with Acute Myocardial Infarction by Age Groups.

Age (years)	Patients Admitted to CCU n (%)	Patients Not Admitted to CCU n (%)	Relative Risk CCU Nonadmission (95% CI)
40–74	557 (90)	63 (10)	1.0
75–79	69 (79)	18 (21)	2.09 (1.16,3.37)
80–84	50 (81)	12 (19)	2.18 (1.09,4.36)
85+	27 (66)	14 (34)	4.25 (2.10,8.59)

Relative risks are adjusted for admitting hospital and compare rates of CCU nonadmission for older age groups to those of patients in the 40–74 year age group.

ticipate in the study. Patients were enrolled during a 16-month period ending in August 1981. For the present study, we excluded all data collected during the experimental phase of our previous clinical trial of CCU use.[9]

Data Collection

Clinical data, cardiac-enzyme tests, and electrocardiograms (ECGs) were obtained at the time of entry for all consenting patients. Additional clinical data—including clinical records, follow-up ECGs, and cardiac-enzyme tests—were obtained for both hospitalized and nonhospitalized patients at 48 hours after admission. Mortality was determined from study inclusion through follow-up, including the index hospitalization for hospitalized patients. The overall follow-up rate was 89 percent.

Data Analysis

Study patients were stratified by age into a younger group (40–74 years) and an older group (75 years of age or older). Final diagnoses were assigned for each patient based on blinded review of the serial ECGs, results of the cardiac-enzyme tests, and the ER and hospital discharge records. Final "true" diagnoses of acute myocardial infarction or angina pectoris were based on clinical presentation, ECGs, and cardiac-enzyme tests using the World Health Organization[11] and New York Heart Association criteria.[12] We assigned final diagnoses on the basis of the patient's ER clinical presentation. Patients admitted to the hospital with other diagnoses, who subsequently had myocardial infarctions in hospital, were analyzed according to their initial diagnosis. For patients with acute myocardial infarction, clinical severity was stratified by Killip class at presentation.[13]

The probability of not being admitted to the CCU (i.e. CCU nonadmission) was the primary end-point of this study. For the purposes of this study, CCU care and ICU care are considered equivalent. "Step-down" Units were not included as CCU care because such units represent a less intensive standard of care. At the time of this study, CCU care was the accepted standard of care in the United States for patients with acute ischemic heart disease.[14] Hence, CCU nonadmission is considered to be an adverse event for patients with acute myocardial infarction.

Statistical Methods

All hypothesis tests were two-tailed. The hypothesis that physicians' CCU admitting practices were related to patient age (Table 1) was tested using a logistic regression model predicting the probability that the patient was not admitted to the CCU. Separate models were analyzed for each group of final verified diagnoses (acute myocardial infarction, angina pectoris and no acute ischemic heart disease). The factors included in each model were: age, gender, the hospital where the patient was evaluated (to control for differences in admitting rates among hospitals), and the occupancy level of the CCU at the time of the patient's admission. Patients over 75 years of age with acute myocardial infarction were also stratified by five-year age intervals (Table 2) and a similar logistic regression model was used to calculate age-specific relative risks for CCU nonadmission.[15]

The test for linear trends in proportions[16] was used to examine trends in CCU nonadmission rates for patients with myocardial infarction when stratified by Killip Class (Table 3). To test the hypothesis that differences in physicians' CCU admitting practices for patients with acute myocardial infarction were related to differences in clinical presentation between older and younger patients (Table 4), we used a multivariate logistic regression model predicting CCU nonadmission. The factors included in the model were: age, gender, the hospital where the patient was evaluated, presenting features, and separate interaction terms to detect relationships between age and each of the presenting features. The interaction terms were used to examine whether advanced age and the presence of a particular clinical sign or symptom had a significant impact on CCU nonadmission.

Tests for the observed differences in CCU nonadmission rates for patients on the basis of the ER physician's admitting diagnosis (Table 5) were performed using the Mantel-Haenszel Chi-square procedure to control for differences in hospital admitting rates.[17]

To examine the relationship between mortality and CCU nonadmission for older versus younger patients with acute myocardial infarction, we used a logistic regression model predicting mortality. The factors included in the model were: age, gender, the hospital where the patient was evaluated, CCU nonadmission and an interaction term for the relationship between age and CCU nonadmission. The interaction term was used to examine whether CCU nonadmission had a significant impact on the mortality of elderly patients with acute infarctions.

Results

The average sample size for the six hospitals was 704 patients and ranged from 453 to 938 patients. By hospital type, 1,505 patients were seen in the urban medical school teaching hospitals, 1,309 in the medical school-affiliated teaching hospitals in smaller cities, and 1,409 in the rural non-teaching hospitals.

The study included 3,225 patients (76 percent) ages 40 to 74 years, and 998 patients (24 percent) who were 75 years of age and older. The mean age was 58.9 years among the younger group of patients, and 81.0 years among the older patients. Male patients constituted 58 percent of the younger patients versus 41 percent of the older patients. Of all study patients presenting to the ER, 810 patients (19 percent) had acute myocardial infarction, 710 patients (17 percent) had angina pectoris, and 2,703 (64 percent) proved to have neither form of acute ischemia. No significant differences were found between younger and older study patients in the proportion of patients with acute myocardial infarction (19 percent in each group) or angina pectoris (18 percent vs 14 percent, respectively).

TABLE 3—Distribution of Acute Myocardial Infarctions and CCU Nonadmission by Presenting Killip Classes among Younger and Older Emergency Room Patients (N = 810)

	Patients with Acute Myocardial Infarction n (%)		CCU Nonadmission for Patients in Each Killip Class n (%)	
Killip Class	Age 40–74 (n = 620)	Age ≥ 75* (n = 190)	Age 40–74 (n = 63)	Age ≥ 75** (n = 44)
1	429 (69)	80 (42)	48 (11)	14 (18)
2	107 (17)	47 (25)	9 (8)	11 (23)
3	62 (10)	48 (25)	5 (8)	19 (40)
4	22 (4)	15 (8)	1 (5)	0 (0)

*$p < 0.0001$ for trend of increased proportion of older patients with higher Killip classes.
**$p < 0.0001$ for trend of decreased CCU admission of older patients with higher Killip classes.
Percent of patients not admitted to the CCU are calculated relative to the numbers of patients in each age group within that Killip class.

Although not presented here, the triage dispositions of patients 65–74 years of age did not significantly differ from those of younger patients, reinforcing the inclusion of this group with the middle-aged group. Age 75 years was selected as best representing a truly elderly group of patients, as noted in other discussions of health care for the elderly.[1,5]

Among ER patients who proved to have acute myocardial infarction (Table 1), CCU nonadmission occurred 2.50 times more frequently for patients 75 years of age or older than for patients 40 to 74 years of age (95% CI = 1.64, 3.85). Examination of the differences in CCU nonadmission rates among ER patients with acute myocardial infarction, stratified by five-year age intervals for older patients, shows that the likelihood of CCU nonadmission increases with advancing age (Table 2).

Among ER patients with angina pectoris (Table 1), the probability of CCU nonadmission for older patients compared to younger patients was less evident (RR 1.45, 95 percent CI = 0.98, 2.13). For patients without acute ischemic heart disease, CCU nonadmission rates for older and younger ER patients were not significantly different, although older ER patients were significantly less likely to be discharged to home from the ER than younger patients (RR 0.26, 95 percent CI = 0.21, 0.32).

Differences in the clinical severity of acute myocardial infarctions by Killip classes at the time of presentation for older and younger patients are shown in the first set of columns in Table 3. A clear trend towards an increasing proportion of more serious infarctions in older patients is seen. The second set of columns in Table 3 shows the differences in CCU nonadmission rates for older versus younger patients in each presenting Killip classes. For all presenting Killip classes, except Killip class 4, CCU nonadmission was more likely among older patients than younger patients. Among study patients with Killip class 4 myocardial infarctions, defined as presentation with cardiogenic shock, only one patient was not admitted to the CCU. The apparent trend towards increasing CCU nonadmission rates with advancing Killip class for older patients with Killip class 1, 2, and 3 myocardial infarctions is also clear.

To test the hypothesis that older patients with infarction were admitted to the CCU less frequently than younger patients due to less clearly diagnostic presenting signs and symptoms, we examined gender, ECG findings, and presenting complaints of chest pain, dyspnea, or other symptoms (Table 4). Two ECG findings indicative of acute ischemic cardiac disease, T-Wave peaking or inversion and ST-segment elevation or depression greater than 1 mm, increased the likelihood of CCU admission. Aside from age, only presentation with dyspnea and no chest pain predicted a decreased likelihood of CCU admission. We found no significant interactions in the regression equation between advanced age and any other presenting feature. Thus, older patients with any specific presenting feature were neither more nor less likely to be admitted to the CCU than younger patients with similar presentations. Furthermore, controlling for the effects of all of

TABLE 4—Multivariate Logistic Model of Presenting Features Associated with Nonadmission to the Coronary Care Unit Admission of Emergency Room Patients with Acute Myocardial Infarction (N = 810)

Presenting Feature (n)	Relative Risk for Nonadmission to the CCU RR (95% CI)
Age ≥ 75 (190)	2.4 (1.5, 3.8)
Male (503)	0.9 (0.5, 1.4)
Chest Pain in the ER (693)	1.6 (0.7, 4.0)
Both Dyspnea and Chest Pain (199)	1.1 (0.6, 1.9)
Dyspnea and No Chest Pain (78)	4.5 (1.8, 11.1)
No Dyspnea and No Chest Pain (56)	0.4 (0.1, 1.3)
ECG: T-Waves Peaked or Inverted (540)	0.5 (0.3, 0.7)
ECG: ST Elevation or Depression (681)	0.4 (0.2, 0.6)

No statistically significant interactions were found between age and other presenting features. NS—feature is not a significant predictor of difference in triage disposition at the $p = 0.05$ level.

TABLE 5—Triage Disposition for Younger and Older Emergency Room Patients by Emergency Room Physician's Admitting Diagnosis (N = 4,223)

	Emergency Room Patients with Each Diagnosis n		Patients with Each Diagnosis Not Admitted to the CCU n (%)	
Admitting Diagnosis	Age 40–74 (n = 3,225)	Age ≥ 75 (n = 998)	Age 40–74	Age ≥75
"Myocardial Infarction"	225	59	5 (2)	8 (14)
"Rule-out Myocardial Infarction"	1,116	294	223 (20)	79 (27)
All Other Diagnoses	1,854	645	1,502 (81)	497 (77)

Percentages of patients not admitted to the CCU are calculated relative to the numbers of patients in each age group with the diagnosis.

the presenting features on CCU nonadmission in a multivariate logistic model had no significant impact on the risk of age as an independent negative predictor of CCU admission.

To provide further support for the hypothesis that differences in triage disposition of elderly patients did not result simply from misdiagnosis, we examined the admitting diagnoses of the ER physicians to find whether differences in CCU admissions occurred for older and younger patients even when the ER physician believed the patient to have acute ischemic heart disease (Table 5). Among all ER patients with an admitting diagnosis of "myocardial infarction," 86 percent of patients 75 years or older were admitted to the CCU, compared with 98 percent of younger patients. Expressed as a relative risk, these older patients were 7.1 times more likely not to receive CCU care (95% CI = 2.1, 25.0). For ER patients with an admitting diagnosis of "rule-out myocardial infarction," older patients were 1.5 times more likely not to receive CCU care (95% CI = 1.1, 2.1). Further, upon review of the available 28 ER medical records for the 44 older patients with acute infarction not admitted to the CCU, there was no documentation that the patients themselves had requested a less intense level of care, or that they had impaired mental or social function.

Finally, did differences in triage disposition for older and younger patients have a measurable impact on mortality? Among all patients with diagnosed acute infarctions, mortality was greater for older patients than younger patients (29 vs 12 percent), but was similar among older patients admitted to the CCU versus those admitted to the wards or sent home (RR 1.12, 95% CI = 0.38, 3.7).

Discussion

The results of this study suggest that advanced age has an impact on the CCU admitting decisions made by physicians, most evident among ER patients with acute myocardial infarctions. None of the hospitals participating in this study had an explicit policy of rationing intensive care to elderly patients and lower CCU admission rates for older patients with acute infarction was not related to the occupancy level of the CCU. The observed difference in CCU admission rates for older patients with acute infarction did not result simply from diagnostic misclassification in the ER. Nevertheless, our data do not allow us to directly observe the reasoning or motivation underlying the triage decision.

Physicians' awareness that a study of CCU admitting practices was taking place may have affected our results even though physicians were not aware of the study hypothesis. Differences in the triage disposition of older and younger patients might have occurred if physicians improved their diagnostic accuracy more in younger patients than in older patients. However, this fails to explain why physicians, even when they had made a diagnosis of acute ischemic heart disease, as indicated by their admitting diagnosis of "myocardial infarction" or "rule-out myocardial infarction," were still less likely to admit an older patient to the CCU.

This study was primarily designed to examine differences in CCU admitting practices, and despite its size, its power to detect small differences in mortality and other outcomes among different subgroups of patients is limited. This is reflected by the fairly wide confidence intervals for the relative risk of mortality in older patients based on CCU admission. Because Killip classes are correlated with mortality,[13] the observation that among patients not admitted to the CCU, older patients had higher Killip classes of infarctions than younger patients suggests that differences in mortality may indeed exist.

Several factors not included in our data set may influence the ER physicians' decision not to admit elderly patients to the CCU. First, physicians may believe that CCU care for elderly patients is not cost-effective, particularly since unequivocal evidence demonstrating the efficacy of CCUs is lacking[18,19] and there is some evidence that medical intensive care is probably overutilized in the United States.[19] However, the differences in triage disposition we describe occur among patients with acute myocardial infarction, for whom CCU care is the present standard of care regardless of age. Thus, the notion that the physicians in our study were somehow utilizing CCU care more effectively for older than younger patients does not provide an appropriate explanation for our findings.

A second concern is that CCU care may be potentially harmful for older patients, because of the association between age and an increased likelihood of iatrogenic complications.[20] Thus, physicians may intentionally restrict CCU care for older patients for compassionate reasons.

Third, mentally competent patients, by exercising their acknowledged rights of self-determination, may themselves influence treatment decisions by refusing more intense levels of care.[21] However, review of available ER medical records for older patients with acute infarction in our study, who were not admitted to the CCU, yielded no evidence that patients themselves had influenced the admitting decision.

Fourth, socioeconomic factors may affect the level of intensity of care given to patients. For example, marital and insurance status have recently been shown to influence the choice of therapy for patients with lung cancer.[22]

Finally, it has been shown that the patient's capacity to interact meaningfully with others in established social roles influences physicians' decision making in the management of critically ill patients.[4] Thus, physicians might intentionally restrict CCU admission for elderly patients with dementia or severe disability. In our study, all participating patients were able to give informed consent and provide necessary information to the research assistants, so it is unlikely that dementia among older patients could account for the observed differences in CCU admission. Moreover, review of the available charts of elderly patients not admitted to the CCU, despite having admitting diagnoses of "myocardial infarction" or "rule-out myocardial infarction," yielded no evidence that such considerations played a role in this decision.

Several previous studies have examined the influence of age on ICU admission practices. In a study conducted at an urban hospital, it was noted that, although 10 percent of the general population age 75 years and older reside in nursing homes, only 5 percent of the ICU/CCU patients were nursing home residents.[23] In another study, the age distribution of patients receiving respiratory intensive care was found to be similar to the age distribution of the general hospital population, except for patients over 70 years of age, among whom there were fewer ICU admissions.[24] In a third study, examining selected patients admitted to the ICU and the general medical wards at the same hospital, no difference was found between the age distributions of ward patients and ICU patients, although there was a borderline significant higher severity of illness for older medical ward patients.[25] Overall, these studies, which looked at patterns of care at single institutions, provide indirect evidence of selective triage to ICU care for older patients.

This prospective multicenter study, following patients from their presentation in the ER, provides more direct evidence of restrictive use of intensive care for the elderly. If age influences the likelihood of ICU admission, then studying ICU patients alone, or even ICU along with ward patients, will introduce inclusion bias in study subject selection. To properly interpret data concerning the benefit or access to ICU care for elderly patients, it is important to study the "denominator" group of all patients seen in the emergency room who were candidates for ICU or ward admission, rather than "numerator" patients in one or two specific sites within the hospital.

The fact that the data for this study were collected between 1979 and 1981 might be considered a limitation to the usefulness of our findings. However, an advantage of these data is that they were collected prior to the advent of Medicare's prospective payment system. Provision of intensive care, a costly part of hospital care, is susceptible to PPS incentives,[4] which might confound a study of physician or CCU admission practices done more recently.

The data on which our findings are based also precede the widespread use of thrombolytic therapy for patients with acute myocardial infarctions. Most randomized trials of thrombolysis have excluded patients over the age of 70 because of fear of increased complications in the elderly.[26] If the availability of thrombolytic therapy creates an incentive to preferentially admit to the CCU those patients who receive thrombolysis, this may create another bias against CCU admission in elderly patients, particularly when limited CCU resources are available.[19] As hospital payment policy evolves and ongoing and planned trials of thrombolytic therapy that include aged patients are completed, these influences may be mitigated.

We have presented data suggesting that advanced age has an impact on the triage disposition of patients presenting with suspected acute ischemic heart disease that is not explained by differences in severity of illness or varying clinical presentations among older patients. While our findings are not definitive proof, they suggest that physicians may intentionally restrict access to coronary care for elderly patients. These findings should prompt further investigations to find consistent or countervailing evidence for elderly patients in other intensive care settings and with other conditions. ☐

Acknowledgments

Supported in part by grants R01-HS02068 and R01-HS05549 from the National Center for Health Services Research and by training grant LM 7044 from the National Library of Medicine, Bethesda, MD. Dr. Selker was an American College of Physicians Teaching and Research Scholar during the time this research was conducted.

We are indebted to Joni R. Beshansky, RN, Robert L. Jayes, MD, Bruce D. McCarthy, MD, Stephen G. Pauker, MD, and Joellen M. Schildkraut, PhD, for their suggestions during the course of this study. We wish to thank Elliott Fisher, MD, MPH, Harry R. Kimball, MD, Herbert J. Levine, MD, Deeb N. Salem, MD, and Mark Wenneker, MD, for their helpful comments on this manuscript.

References

1. Scitovsky AA, Capron AM. Medical care at the end of life: The interaction of economics and ethics. *Annu Rev Public Health.* 1986;7:59–75.
2. Schneider EL, Brody JA. Aging, natural death, and the compression of morbidity: Another view. *N Engl J Med.* 1983; 309:854–855.
3. National Center For Health Statistics. Vital and Health Statistics. Series 3. No. 23. Changing mortality patterns, health services utilization, and health care expenditures. United States, 1978-2003. Hyattsville, MD: Public Health Service, 1983. DHHS pub. no. PHS-83-1407.
4. Mulley AG. The allocation of resources for medical intensive care. In: Securing Access to Health Care. Vol. 3. Washington, DC: The President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research, 1983;285–311.
5. Callahan DJ. Setting Limits. New York: Simon and Schuster, 1987.
6. Schwartz WB, Aaron HJ. A tough choice on health care costs. New York Times April 6, 1988; CXXXVII:A23.
7. Leaf A. Medicine and the aged. *N Engl J Med.* 1980;302:938–942.
8. Knaus WA, Wagner DP. Intensive treatment for the elderly [letter]. *JAMA.* 1982; 247:3185–3186.
9. Pozen MW, D'Agostino RA, Selker HP, et al. A predictive instrument to improve coronary-care-unit admission practices in

acute ischemic heart disease. *N Engl J Med.* 1984;310:1273–1278.

10. Vand Der Does E, Lubsen J, Pool J, et al. Acute coronary events in a general practice: Objectives and design of the imminent myocardial infarction Rotterdam study. *Heart Bull.* 1976;7:91–98.
11. Gillum RF, Fortmann SP, Prineas RJ, et al. International diagnostic criteria for acute myocardial infarction and stroke. *Am Heart J.* 1984;108:150–158.
12. Criteria Committee of the New York Heart Association: Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels, 8th Ed. New York: New York Heart Association, 1979;288–290.
13. Killip R, Kimball JT. Treatment of myocardial infarction in a coronary care unit: A two-year experience with 250 patients. *Am J Cardiol.* 1967;20:457–464.
14. Siegel DM. The high cost of medical technology: Getting at the heart of the matter. *Med Care.* 1987;25:979–987.
15. Lemeshow S, Hosmer DW Jr. Estimating odds ratios with categorically scaled covariates in multiple logistic regression analysis. *Am J Epidemiol.* 1984;2:147–151.
16. Snedecor GW, Corcoran WG. Statistical Methods, 7th Ed. Ames, IA: Iowa State University Press, 1980;246–248.
17. Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. *JNCI.* 1959;22:719–748.
18. Bloom BS, Peterson OL. Patient needs and medical-care planning: The coronary-care-unit as a model. *N Engl J Med.* 1974; 290:1171–1177.
19. Selker HP, Griffth JL, Dorey FJ, D'Agostino RB. How do physicians adapt when the coronary care units is full? A prospective multicenter study. *JAMA.* 1987;257:1181–1185.
20. Steel K, Gertman PM, Crescenzi BS, et al. Iatrogenic illness on a general medical service at a university hospital. *N Engl J Med.* 1981;304:638–642.
21. Bellamy PE, Oye RK. Admitting elderly patients to the ICU: Dilemmas and solutions. *Geriatrics.* 1987;42:61–68.
22. Greenberg ER, Chute CG, Stukel TA, et al. Social and economic factors in the choice of lung cancer treatment: A population-based study in two rural states. *N Engl J Med.* 1988;318:612–617.
23. Campion EW, Mulley AG, Goldstein RL, et al. Medical intensive care for the elderly: A study of current use, costs, and outcomes. *JAMA.* 1981;246:2052–2056.
24. McLean RF, McIntosh JD, Kung GY, et al. Outcomes of respiratory care for the elderly. *Crit Care Med.* 1985;13:625–629.
25. McClish DK, Powell SP, Montenegro H. The impact of age on utilization of intensive care resources. *J Am Geriatr Soc.* 1987; 35:983–988.
26. Wasserman AG, Ross AM. Patient selection for thrombolytic therapy. *Am J Cardiol.* 1989;64:17B–21B.

Migrant Farm Workers in Need of Better Health Care

The Migrant Clinicians Network recently released the results of *Migrant Health Status: Profile of a Population With Complex Health Problems*, the first comprehensive investigation into health conditions for migrant and seasonal farm workers. Specific to those farm workers who migrate within the midwestern migratory stream, the study reveals an overall health status with higher rates of infectious disease than in the general population.

The results from this study are shocking. The demographic patterns, socioeconomic conditions, life-style characteristics, and disease categories reflect an agrarian society in a Third World country caught in an infectious disease cycle. Factors such as poverty, malnutrition, infectious and parasitic diseases, poor education, a young population, and poor housing equate to a highly vulnerable population in need of resources. Clearly, the migrant population is at greater risk and suffers more problems than the general population. The results of this research demonstrate the need for more service, care, and treatment. The need for developing a health policy and research agenda for migrant farm workers in this decade is evident.

The Migrant Clinicians Network (MCN) is a multidisciplinary organization of all health and human service professionals who work with migrant and seasonal farmworkers. The network was established in 1984 to identify and address clinical issues affecting the health status of the nation's migrant farmworker population. MCN comprises migrant clinicians from 41 states and Puerto Rico, representing a wide range of professional disciplines and areas of expertise. The Network serves as a clinical forum for migrant and seasonal farmworker health issues.

The health status study was conducted under the direction of G. E. Alan Dever, PhD, of Mercer University. For a copy of the executive summary, study monograph, or complete report contact the Migrant Clinicians Network, 2512 South IH35, Suite 220, Austin, TX 78704. Tel: 512/447-0770.

Comparison of Subjective Ratings of Function with Observed Functional Ability of Frail Older Persons

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ABSTRACT

Background. Important clinical decisions often hinge on patients' functional status. Previous studies have shown disagreement among sources of ratings of patients' functional status. This study compared patient self-ratings, family member ratings, and physician ratings of patient function to performance-based functional testing criteria.

Methods. Five activities of daily living of 73 older patients were studied at admission to a rehabilitation unit following discharge from an acute care community hospital. Data were collected from patients, family members, and physicians and were compared with performance-based function testing.

Results. Patient ratings were significantly more accurate than physician ratings for walking, transferring, and telephoning. Patients were significantly more accurate than family members for rating walking and telephoning, but patients were not significantly more accurate than family members or physicians for rating eating or dressing.

Conclusions. We conclude that decisions about patients' functional level should be based on performance testing. If performance testing is unavailable, patients' own ratings are most accurate, followed by family ratings. Physicians' ratings are least accurate. (*Am J Public Health.* 1991;81:1127–1130)

Introduction

Functional assessment of elderly patients has been a focus of research for more than 25 years.[1] Its importance is demonstrated by the one fifth of persons aged 65 and older in the United States who are unable to perform at least one measure of activities of daily living (ADL).[2] The complexity of functional measurement is demonstrated by more than 40 published ADL scales.[3] Most ADL scales use patient self-report to rate performance of tasks. Recently investigators have compared ADLs measured by an assessment instrument or geriatric evaluation to patient self-report, family report,[4] or physician report[5] and found that different sources of ADL rating may not agree and therefore are not interchangeable.

A recent review by Guralnik and colleagues[6] makes a strong case for the value of performance-based ADL measures. Elderly patients who experience an acute illness or injury often have decreased function requiring decisions ranging from assistance with personal care to permanent nursing home placement. During hospitalization patients may be unable to assess their own functional status because their activities may be medically limited. Thus, accurate assessments of function are needed to ensure appropriate recommendations. Because sources of ADL ratings may disagree, comparisons of different rating sources with objective measures of performance are important to characterize the accuracy of various rating sources.

This study was designed to evaluate performance-based ADL testing in a community hospital rehabilitation unit. Unlike previous studies, we compared the accuracy of self-ratings, family ratings, and physician ratings to performance-based measures.

Methods

Study Sample

Seventy-three patients aged 60 years and older were studied between January 1987 and March 1989. These patients had been hospitalized at the Baptist Memorial Hospital, a large community hospital, for acute medical or surgical problems and required additional rehabilitation.[7] Patients were eligible for enrollment in the study if they agreed to participate, signed informed consent, were cognitively able to participate (no more than four errors on the Short Portable Mental Status Questionnaire),[8] were not aphasic, and had a family member currently involved in their care. This was defined as a relative who visited the patient during the acute care hospital stay and was willing to assume responsibilities for assistance when the patient was discharged.

Measurement

Although the number of days of the acute hospital stay was not recorded, all

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This paper was submitted to the journal May 25, 1990, and accepted with revisions March 15, 1991.

TABLE 1—Demographic and Functional Health Data (N = 73)

Variable	Data
Age (years)	
Mean ± SD	77.3 ± 7.9
Range	61–98
Sex (% female)	62.5
Race (% white)	87.7
Marital status (% married)	31.5
Baseline ADLs (% independent)[a]	
Walking	42.5
Transferring	52.8
Telephoning	77.5
Eating	93.1
Dressing	77.5

[a] Observed performance test data.

patients enrolled in the study were medically stable and ready for rehabilitation. As part of the initial evaluation at transfer to geriatric rehabilitation (usually the same day) patients were first asked to rate their abilities to walk, dress, eat, transfer, and use the telephone, using a standard format from the Framingham Disability Study.[9] Five performance levels were possible: (1) independence, (2) requires use of equipment, (3) requires assistance of another person, (4) requires assistance of another person and equipment, or (5) unable to perform. These same abilities were rated separately by the caregiving family member and by the patient's personal physician. At admission patients were also asked to perform five tests which corresponded to the five rated abilities: (1) walking; walk 25 feet; (2) dressing: button a shirt; (3) eating: transfer dried beans with a spoon from one dish to another; (4) transferring: transfer from bed to chair; and (5) telephoning: make a telephone call.

Interviews and performance-based testing were conducted by a nurse clinician or by one of two physician research fellows. Family members were interviewed at the rehabilitation unit or by telephone. All interviews with patients' personal physicians were made by telephone. Interviews with family members and physicians were made within 48 hours of the patient interview or considered unobtainable. Demographic information was collected from patient's medical records.

Patient interviews and observational protocols were completed for 73 patients. Sixty-four (88%) family member and 49 (67%) physician interviews were completed within 48 hours of the time patient data were collected. Complete observational, patient, family, and physician data collection protocols were executed for 41 (56%) patients. Data on observed ADL, age, sex, race, and marital status were analyzed to detect potential bias caused by some physicians' nonparticipation. No statistically significant differences in these variables were found between patients whose physicians participated and those whose physicians did not.

Data Analysis

Data were analyzed using the SPSS^x system of computer programs, including CONDESCRIPTIVE for descriptive statistics, CROSSTAB for truth tables, and PEARSON CORR for correlations.[10] The χ-square test was used to determine the independence of ratings vs the performance test criterion. Differences between these χ-square tests were used to test for differences in the agreements of ratings with the criterion. In all cases, $P<.05$ was used as the criterion of statistical significance. Significance levels were adjusted for multiple comparisons when appropriate through use of the Bonferroni method.[11]

For analysis of accuracy of ADL ratings the five levels of ability for each ADL were dichotomized as independent vs dependent. Independence was defined as performance of the ADL without assistance from another person, and dependence was defined as nonperformance or need for the assistance of another person to perform the ADL.

Actual performance ability, assessed by direct observational testing, served as the criterion of performance of an ADL. Criteria for evaluating diagnostic tests (including sensitivity, specificity, predictive value of a positive test, and predictive value of a negative test) were adopted to evaluate the accuracy of ratings by use of the analogy whereby functional dependence was equated with the presence of a disease condition, i.e., a positive test. Thus, sensitivity was defined as the number of patients rated dependent who also were observed to be dependent divided by the number observed to be dependent. Specificity was defined as the number of patients rated not dependent who also were observed to be not dependent divided by the number observed to be not dependent. Predictive value of rated positive was defined as the number rated and observed to be dependent divided by the number rated dependent. Predictive value of rated negative was defined as the number rated and observed to be not dependent divided by the number rated not dependent.

Results

Demographic and functional health data for the 73 study patients are presented in Table 1. Patients were transferred to rehabilitation with diagnoses representing all major rehabilitation groups. Twenty-four percent had a primary diagnosis of stroke, 17% had experienced fractures (of these 50% were hip fractures), 48% had a primary medical diagnosis, and 11% had other problems.

To investigate overall rater performance in accurately documenting patient ADL status, zero-order correlations of patient, family, and physician ratings for each ADL compared to observed performance are presented as phi coefficients in Table 2. For walking, the ratings of patient, family, and physician were all significantly correlated to observed performance. For transferring, telephoning, and dressing, both patient and family ratings were significantly correlated to observed performance. For eating, only patient ratings were significantly related to observed performance. Thus, patient ratings were significantly correlated to performance data for five of five ADL, family ratings for four of five, and physician ratings for one of five.

Other results comparing observed performance to patient, family, and physician ratings are also shown in Table 2. For walking and transferring, physician ratings have low specificity (walking=45%, transferring=38%) due to underestimation of the number of independent patients. Patient self-ratings ($P<.005$) and family ratings ($P<.005$) were both significantly more highly correlated to observed performance than were physician ratings for walking and transferring. In addition, patient self-ratings for walking were significantly more highly correlated to observed performance than were family ratings ($P<.005$).

For telephone use, physician ratings had low sensitivity (25%) because physicians rated too few patients as unable to use the telephone. Family raters also had difficulty with rating telephone use, and their underestimation of dependency was documented by the family raters' low predictive value of rated positive (42%). Patient self-ratings ($P<.005$) and family ratings ($P<.05$) were both significantly more highly correlated to observed performance than physician ratings for telephone use. As with walking, patient self-ratings of telephone use were also significantly more highly correlated to observed performance than family ratings were ($P<.005$).

For eating, patient, family, and physician too often rated patients as dependent, resulting in poor predictive values of rated positive. In addition, physician ratings lacked sensitivity, not detecting which patients were dependent in eating. All raters lacked specificity and showed poor predictive values of rated positive for dressing, chiefly because of underrating independent dressers.

Discussion

When an elderly patient has an acute illness or accident, decisions about the level of care required at hospital discharge can permanently alter the patient's living arrangement. Questions arise concerning need for postdischarge services such as home health care. Although on the one hand it is advisable to avoid inappropriate institutional placements, on the other hand patients living in the community who are too functionally dependent to care for themselves pose a burden for themselves and others.

How best to evaluate functional level is a question with which researchers have long struggled. Self-reported ADLs may include potential that the patient has not yet regained or represent performance that the patient may be unable to attain at the time of observation because of temporary physical status or environmental limitations. Our experience with performance testing supports the opinions of Guralnik et al.[6] that performance testing can be easily administered, is not overly time consuming, is well received by patients, and results in no harm to patients. Although we did not use the same tests, our methods for measuring function were similar to those described by Guralnik and colleagues. Also, the general agreement of patient and family ratings with observed function data validates performance testing.

Our data showed that patients and, to a lesser degree, family members had good knowledge of the patient's ability to walk, transfer, use the telephone, and dress. Also, patient ratings of eating ability were fairly accurate. By type of function, the predictive value of rated positive was highest for patient and family for the lower extremity skills, i.e., walking and transferring. For upper extremity skills the predictive value of rated positive was uniformly lower.

TABLE 2—Estimates of Predictive Accuracy

			Predictive Value of Rated			
ADL and Rater	Sensitivity, %	Specificity, %	Positive, %	Negative, %	Correct, %	Phi Coefficients[a]
Walking						
Patient	90.5	90.3	92.7	87.5	90.4	0.80*
Family	83.3	67.9	76.9	76.0	76.6	0.52*
Physician	82.8	45.0	68.6	64.3	67.3	0.30**
Transferring						
Patient	76.5	81.6	78.8	79.5	79.2	0.58*
Family	90.3	67.6	71.8	88.5	78.5	0.59*
Physician	73.9	38.5	51.5	62.5	55.1	0.13
Telephoning						
Patient	56.2	92.7	69.2	87.9	84.5	0.53*
Family	61.5	78.4	42.1	88.9	75.0	0.35***
Physician	25.0	87.5	33.3	82.4	75.0	0.14
Eating						
Patient	80.0	71.6	17.4	98.0	72.2	0.28**
Family	75.0	71.4	14.3	97.8	71.6	0.23
Physician	40.0	78.6	18.2	91.7	74.5	0.14
Dressing						
Patient	100.0	41.0	23.4	100.0	50.0	0.31****
Family	100.0	35.3	25.0	100.0	46.8	0.30**
Physician	87.5	35.3	21.9	92.3	42.2	0.17

[a] Statistical tests of association between rater and performance data.
*$P < .001$.
**$P < .05$.
***$P < .005$.
****$P < .01$.

Physicians underestimated independence for lower extremity ADLs and overestimated independence of ADLs requiring cognitive skills, such as dressing and telephoning. These findings are in agreement with earlier findings of Pinholt et al.,[5] who found that mental status deficits were underreported in a comparison of ratings of residents, interns, and nurses to functional ratings by geriatricians using assessment instruments. Also, our finding that patient and family ratings were significantly different from each other for walking and telephone use agrees with findings published by Rubenstein et al.,[4] who observed significant ADL report differences between patient and family.[4] In the Rubenstein study, patients and family ratings were compared to ratings by nurses who provided daily patient care. This criterion differs from the criterion in our study, which was performance-based ADL testing. As in Rubinstein's study, our family raters underestimated independence in walking and transferring; however, in contrast to Ruberstein's results, our patient raters had the best overall accuracy. Our study differs from previous studies comparing clinical ADL rating to other rating sources in a fundamental way. Our physician raters were all community hospital based private practitioners, responsible for the patient's daily care during the acute care hospitalization. Most were internists who had a long-term relationship with the patient. This is in contrast to the Rubenstein study, which was set in a Department of Veterans Affairs Hospital,[4] and to the Pinholt study, which was set in a military hospital.[5]

This study represents an attempt to learn about the measurable components of ADLs. Choosing the optimum surrogate measure to use in performance testing is difficult, and some of the differences observed between performance testing and subjective ratings may be due to the fact that the performance measures do not assess all the complex aspects of a given ADL. Variables like walking and transferring can be observed directly. For other ADLs such as dressing and grooming, which require multiple functional and cognitive skills, choosing surrogates is more difficult. For example, our choice of shirt buttoning as a surrogate for dressing may not have been optimal because buttoning lacks representation of lower extremity dressing and does not represent the cognitive processes necessary to select clothing and to don garments in order.

This study has some limitations. Study patients were not representative of the entire population of acute care patients because all had family members involved in their care and all were eligible for rehabilitation. Demented patients may not report their function accurately and were excluded from our study. Our study did not observe continence and toileting, and these ADLs were not included in our ratings. Personal physicians were asked to answer immediately to our inquiry and were not given an opportunity to collect additional information from observations, patients, or family. However, they were the attending physicians during the hospitalization and had ample opportunity to collect these data. All study physicians practiced in a large community hospital in a metropolitan area. These results may not apply to other service settings and may not, for example, represent the situation in nonmetropolitan areas, where physicians might have a lifetime relationship with the patient.

In summary, physicians would be well advised to formulate recommendations that are dependent on function after obtaining objective measures of performance. If performance testing is unavailable, patient self-rating usually serves well for ADLs, especially those related to ambulation. For patients incapable of giving reliable ratings, family rating is the best substitute. If physicians would include questions concerning performance in these areas in their routine history-taking, their knowledge of their patients' performance might be improved. ☐

Acknowledgments

This paper was presented, in part, at the 42nd Annual Meeting of the Gerontological Society of America, November 17 to 20, 1989.

References

1. Katz S, Ford AB, Moskowitz RW, et al. Studies of illness in the aged. *JAMA*. 1963;185:914-919.
2. Leon J, Lair T. Functional status of the noninstitutionalized elderly: estimates of ADL and IADL difficulties. *National Medical Expenditure Survey Research Findings 4*. Rockville, Md: Agency for Health Care Policy and Research publication (PHS) 90-3462. Public Health Service; 1990. US Dept of Health and Human Services.
3. Feinstein AR, Josephy BR, Wells CK. Scientific and clinical problems in indexes of functional disability. *Ann Intern Med*. 1986;105:413–420.
4. Rubenstein LZ, Schairer C, Weiland GD, Kane R. Systematic biases in functional status assessment of elderly adults: effects of different data sources. *J Gerontol*. 1984;39:686–691.
5. Pinholt EM, Kroenk K, Hanly JF, Kussman MJ, Twyman PL, Carpenter JL. Functional assessment of the elderly: a comparison of standard instruments with clinical judgment. *Arch Intern Med*. 1987; 147:484–488.
6. Guralnik JM, Branch LG, Cummings SR, Curb JD. Physical performance measures in aging research. *J Gerontol*. 1989;44: M141–M146.
7. Applegate WB, Akins D, Vanderzwaag R, et al. A geriatric rehabilitation and assessment unit in a community hospital. *J Am Geriatr Soc*. 1983;31:206–210.
8. Pfeiffer E. A Short Portable Mental Status Questionnaire for the assessment of organic brain deficit in elderly patients. *J Am Geriatr Soc*. 1975;23:433–441.
9. Jette AM, Branch LG. The Framingham disability study, II: physical disability among the aging. *Am J Public Health*. 1981;71:1211–1216.
10. SPSS, Inc. *SPSS^x User's Guide*. 2nd ed. New York, NY: McGraw-Hill; 1986.
11. Ingelfinger JA, Mosteller F, Thibodeau LA, Ware JH. Biostatistics in Clinical Medicine. New York, NY: Macmillan; 1983.

Call for Abstracts for APHA Late-Breaker Epidemiology Exchange Session

The Epidemiology Section will sponsor a Late-Breaker Epidemiology Exchange on Wednesday, November 13, 1991, at APHA's Annual Meeting in Atlanta. The exchange will provide a forum for presentation of investigations, studies, methods, which have been conceived, conducted, and/or concluded recently so that abstracts could not meet the deadline for submission to other epidemiology sessions. Papers submitted should report on work conducted during the last 6 to 12 months.

Abstracts should be limited to 200 words; no special form is required. Abstracts should be submitted to Robert A. Gunn, MD, Division of Field Epidemiology, Epidemiology Program Office, Mailstop C08, Centers for Disease Control, Atlanta, Goergia 30333 (Tel: 404/639-3661; FAX: 404/639-3950, and must be received by October 4, 1991). Receipt of abstract will be acknowledged by postcard and acceptance will be decided by October 9, 1991 (please include a return envelope).

Screening Recommendations for the Elderly

Mark H. Beers, MD, Arlene Fink, PhD, and John C. Beck, MD

ABSTRACT

Background. Studies have documented the potential contributions of preventive health care programs. Yet little is known about which screening tests should be included in public health programs for older persons. This study offers recommendations regarding these tests.

Methods. The recommendations come from synthesizing the findings of the US Preventive Services Task Force, the literature, and the consensus of experts in geriatrics, gerontology, and health policy research. The literature was evaluated to identify methodologically sound studies of the prevalence of selected disorders and benefits and availability of screening procedures for those disorders. Experts from various fields specializing in the care of the elderly formed panels to assist in evaluating the literature and providing further information from gerontological and public health perspectives.

Results. We recommend vision testing for refractive error; inspection of the skin surface for fungal infection and skin cancer, drug eruptions, and xerosis; a history for symptoms of xerosis; audiometric testing for presbycusis; surveys for hearing loss; otoscopic inspection for cerumen impaction; dental examination for caries; measurement of blood pressure for hypertension; and breast examination and mammography for cancer.

Conclusions. Our study suggests that these screening procedures are useful for public health screening programs. More information is needed on the effects of screening services on the health and functioning of older persons. (*Am J Public Health.* 1991;81:1131–1140)

Introduction

The American nation is growing older.[1] During the past two decades, the population over 65 has grown more than twice as quickly as the rest of the US population. In 1986, 41% of the elderly were 75 years of age and older; by the year 2000, half the elderly are expected to be over age 75. The 85-and-over population is growing especially rapidly. This group is expected to be seven times as large by the middle of the next century as it was in 1980. The number of functionally impaired elderly is already large and is expected to grow rapidly.[2] As the population ages and becomes more impaired, concern for the prevention of disease and promotion of good health increases.[3]

Studies have documented the potential contributions of preventive health care programs.[4] Advocates assert that preventive programs would result in a healthier and more industrious older population that could assist in reducing the nation's expenses for medical care and social support and contribute to the gross national product.[5] Investigators have documented a high prevalence of undetected, correctable medical conditions among older adults living in the community.[6] As many as 94% of elderly persons screened in a community program were found to have some positive finding requiring advice or information.[7]

Little information is available, however, regarding the screening tests that should be an integral component of a public health program for older persons. Unlike screening in office practice, public health programs do not focus on treatment but, instead, emphasize referrals and prevention. Many, like the California Preventive Health for the Aged Program, are publicly funded, use trained nurses, and are mobile, thus limiting their use of some screening equipment. Among the considerations for screening services for public health programs would be the accuracy of such tests in detecting prevalent, asymptomatic disease, the availability and appropriateness of use of screening, and the availability of beneficial treatment of the diseases that are discovered.

We synthesized findings from the literature, the US Preventive Services Task Force,[8,9] and experts in geriatrics, gerontology, and health policy research to identify screening services that are appropriate for public health programs conducted by nurses.

Methods

Data Sources

Expert Panels. We identified laboratory screening tests, periodic medical review, hypertension, mental health, dermatological problems, ophthalmological problems, dental problems, and hearing problems as areas of greatest concern. Using these as a starting point, we consulted with experts in geriatric medicine and gerontology to help us select specific topics (e.g., stool guaiacs as a laboratory screening test or glaucoma as an ophthalmological condition).

We assembled 24 members of UCLA's academic faculty and clinical ex-

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This paper was submitted to the journal October 10, 1990, and accepted with revisions March 8, 1991.

perts into eight panels corresponding to the areas listed above. Panel members were selected for their knowledge of preventive health care and of the elderly. Panels ranged in size from four to six members, with at least one physician-member of UCLA's Multicampus Division of Geriatric Medicine and Gerontology and one health services researcher with expertise in geriatrics and consensus methods. Other members were expert in one or more of the study topics.

We adopted a two-round consensus method that has been used successfully to resolve issues of uncertainty in health care.[10,11] During the first round, panelists worked independently and without discussion to complete a written questionnaire. They sent the results for synthesis and then met to discuss the findings. Areas of agreement and disagreement were discussed, with the major emphasis on resolution of disagreements. After discussion, the panelists rerated each question.

Literature Review. Our review included studies that were published in English between 1978 and January 1989. We relied on a selective list of investigations considered by experts to be important in guiding geriatric health care practice rather than striving for a review of all published studies.

Inclusion criteria. The purpose of the literature review was to synthesize current knowledge from methodologically sound studies regarding the incidence and prevalence of selected undetected disorders in the elderly and the effectiveness of screening and early treatment. By screening, we meant any methods of detecting disease, regardless of stage or severity; prevention meant primary and secondary measures. Tertiary prevention was not considered in this project. The methods of screening included physical examination and history; patient questionnaires; instruments such as the sigmoidoscope and ophthalmoscope; and laboratory tests such as fasting glucose and thyroid function tests.

Exclusion criteria. We did not review essays or methodologic studies (e.g., to determine the psychometric properties of a measure of function or mental status), although we examined their reference lists for data sources appropriate to our review. We did not include articles whose main purposes were to investigate the treatments and outcomes of care unrelated to screening. We excluded review articles, except in the case of hypertension, because the results of research had been extensively synthesized.

Sources of information. We identified literature through several mechanisms. All members of the expert panels recommended research studies on screening, and references in those papers were also reviewed. We conducted a MEDLINE search simultaneously.

Abstraction of information. We reviewed all articles suggested by the project members, located through secondary sources, or uncovered by the computerized search (see Appendix A), but we abstracted only those that met our inclusion criteria (see Appendix B). We developed an abstraction form for recording the contents of each article. Ten articles were reviewed by three members of the team to establish the reliability of the abstraction process. We focused on the extent to which the inclusion and exclusion criteria discriminated among articles and whether agreement existed on their contents. When we were satisfied with the form's capability to facilitate consistent results, we each reviewed articles; every fifth article received a second review to ensure the continued uniformity of the abstraction process.

Evaluation of the literature. The literature was evaluated on the basis of the quality of the research design using methods similar to those of the Canadian Task Force on the Periodic Health Examination,[12] the US Preventive Services Task Force,[8] and the RAND/UCLA Variations Study.[13] The best evaluations were assigned to studies with the most rigorous designs (e.g., randomized, controlled trials). Only studies that provided convincing evidence through methodological rigor (e.g., controlled studies) were deemed acceptable.

US Preventive Services Task Force. The results of the US Preventive Services Task Force became available in 1989. The credentials of the Task Force panel members and the methods used to arrive at recommendations are described elsewhere.[8] Our study could not rely solely on the results of the Task Force for three reasons. First, the panelists did not explore all the topics in which we were interested, such as functional assessment. Second, although some Task Force recommendations directly affect older persons, the elderly are not the primary focus, and no geriatricians served on the Task Force's 20-member panel. There are many topics, such as the evaluation of dementia or the prevention of functional impairment, for which the perspective of geriatricians and gerontologists may differ from that of other clinicians and researchers. The third reason for not relying solely on the Task Force is that we were interested in public health programs rather than case management by clinicians. The Task Force addressed some diseases that occur almost exlusively in the elderly, such as glaucoma, and it also made specific recommendations for the elderly regarding hearing impairment and bacteriuria. This information was used as a major source of data in this study.

The Synthesis of Data

Our final recommendation for each topic is based on all data sources from which information was available. We assumed that a screening test should receive the highest recommendation if its use is supported by the literature and the US Preventive Services Task Force, it is not currently used appropriately, and it can effectively uncover prevalent, undetected disease for which beneficial treatment is available.

Throughout this project, we used the risk-benefit definition of appropriateness, meaning that a screen is appropriate when its potential benefit outweighs its potential risks. We based our assessment on the effectiveness of screening tests, i.e., the benefit for a given medical problem under average conditions of use.[14]

In this project, available meant that no barriers (such as lack of coverage by Medicare or scarcity of personnel or equipment) existed to prevent patients from receiving a test or treatment. We also included attention to compliance in our definition of availability because patients might not avail themselves of tests or treatments (e.g., hearing aids) if a social stigma or extreme discomfort were associated with them. By prevalent, we meant that the disease or condition occurred in a high enough percentage of elderly persons to warrant screening. We defined undetected disease as a disease or condition that is unknown to the patient or his or her doctor. A test or treatment was determined to be appropriate if those people who should be receiving it were receiving it. We called a treatment beneficial if it could help a substantial proportion of persons with the disease or condition while harming relatively few.

To arrive at our recommendations, we scored each topic (e.g., dental caries, screening for diabetes) on seven items: (1) extent of support from the US Preventive Services Task Force; (2) extent of support from the consultant experts; (3) extent of support from the literature; (4) prevalence of asymptomatic disease associated with the topic; (5) benefit of treatment for the disease; (6) current availability of treatment; and (7) current use of screening. We used a 9-point scale, with 1 = strongly negative (i.e., low prevalence, and little or no support, benefit, or availability), 5 = equivocal (i.e., average, neutral, or uncertain support, prevalence, or availability), and 9 = strongly positive (i.e., high prevalence, and much support, benefit, and availability). The final score for each topic was the mathematical average of ratings on all seven items. The strength of our recommendations is based on this final score according to the following scale. 9 = strongly recommended, 8 = recommended, 7 = recommended with reservation, 6 = equivocal, 5 = equivocal, 4 = equivocal, 3 = not recommended with reservation, 2 = not recommended, 1 = strongly not recommended. For example, a particular screening test might be strongly recommended by the US Task Force (9), and strongly recommended by the UCLA experts (9), although there is little or no literature supporting its use (5). The disease might be common, occurring in over 10% of the population (8). Moderately effective treatment (7) might exist, but the availability of that treatment might be limited due to its high price that is not reimbursed under Medicare and the reluctance of many individuals to undergo the treatment (2). Without new programs to encourage use of this screening procedure, it might be unavailable currently to most elderly persons (8). Such a scenario would receive a final score of 6.9, which we would round off to 7. We support the use of tests that score 7 or better; tests scoring between 4 and 6 are equivocal; and tests scoring between 1 and 3 are not recommended.

Results

Data Sources

Expert Panels. Topics discussed by the panels and their opinions follow.

Hearing

Hearing loss is extremely common in late life, affecting 25% to 35% of older persons, and it increases dramatically with advancing age. Mild hearing loss is more likely to be undetected. The most common causes of hearing loss in late life are presbycusis, cerumen impaction, and noise-induced sensory neuronal hearing loss. The best screening test for presbycusis and noise-induced hearing loss is pure-tone audiometry; the best screening test for cerumen impaction is otoscopic visualization of the auditory canal. Beneficial treatment exists for all three causes of hearing loss, although amplification is sometimes less successful for noise-induced hearing loss than for presbycusis. The use of amplification is limited because of its cost and the social stigma associated with using hearing aids.

Laboratory screening tests

Approximately 2% to 3% of elderly persons have undetected disease that can be diagnosed by routine laboratory tests. The most common undetected diseases (and the best screening methods for detecting each) are anemia (hematocrit or hemoglobin), bacteriuria (urinalysis), hyperlipidemia (serum cholesterol), diabetes (fasting blood sugar), bleeding colonic lesions (stool guaiac), hypothyroidism (TSH), and electrolyte disturbances caused by diuretics and nonsteroidal anti-inflammatory agents (serum electrolytes).

Beneficial treatments exist for hypothyroidism, bleeding colonic lesions (including early cancer), diabetes, and iatrogenic electrolyte disturbances. The benefits of treating anemia depend on the cause. The benefits of treating hypercholesterolemia probably depend on the age of the patient: treating elevated cholesterol in the very old may not be appropriate. Treating asymptomatic bacteriuria is probably beneficial in men but not in women. There is some limitation to the availability of treatment of diabetes due to the cost of medications and monitoring equipment and compliance with diet, medications, and monitoring. Treatment is limited for iatrogenic complications of medication use because physicians are often reluctant to change medical therapy on the advice of nonphysicians or because medical conditions necessitate their continued use. Hyperlipidemia treatment is limited due to the cost and side-effects of medication and compliance with dietary restrictions and exercise programs.

Periodic medical review

Over 50% of the elderly have some undetected disease or condition. The percentage that could be detected through medical history and limited physical examination could not be determined; however, the most common undetected diseases and conditions (and the best screening method for detecting each) that are likely to be found through periodic review are colon cancer (stool guaiacs), breast cancer (breast examination and mammography), coronary artery disease (history), urinary incontinence (history), inappropriate use of medication (medication review), impaired social functioning (screening scales), impaired physical functioning (screening scales), falls (history), and nutritional problems (weight and height). In almost all cases, these tests and procedures are either not currently available to the majority of elderly persons or are not used appropriately at the current time.

Beneficial treatment exists for early colon cancer, early breast cancer, and coronary artery disease. Treatments for the other diseases and conditions are limited at best, and, in some cases, attempts at treatment may be harmful. Treatment availability is limited for urinary incontinence, correcting inappropriate use of medication, and improving poor social and physical functioning, nutritional problems, and falls.

Ophthalmological disorders

Approximately 10% of elderly persons have undetected eye disease or visual impairment, and the prevalence increases with age. The most common diseases and conditions are glaucoma, cataracts, macular degeneration, and uncorrected refractive error; the oldest-old (persons over 85) are most likely to suffer from macular degeneration.

The panel suggested that when screening is performed by nonophthalmologists or nurses, tonometry is the best screening for glaucoma because it is inexpensive, administration takes minimal training, and expensive or bulky equipment is not required. Testing visual acuity is the best screening procedure for all of the other diseases mentioned. Persons with impaired visual acuity should be referred to an eye specialist for futher evaluation. The minimal barriers to obtaining these procedures are mostly due to lack of reimbursement by some Medicaid programs. Visual acuity testing is generally used appropriately, but tonometry is generally not.

The treatment for glaucoma is generally available and beneficial; treatment for cataracts is available and beneficial; treatment for macular degeneration is not beneficial and, if laser treatment is performed inappropriately or incorrectly, may be harmful; treatment for uncorrected refractive error is beneficial and generally available, limited only by cost and reimbursement.

Mental health

Depression can be defined by strict criteria as stated in the DSM-III-R[15] or by a looser definition of affective disorder serious enough to impact on functioning and well-being. Using the strict definition, 1% to 3% of elderly persons suffer from unrecognized depression, but using the looser definition, 7% to 11% are affected. Few data are available on either the best methods for screening for depression in the elderly or the accuracy of the screening. The panel suggested that the eight-item Medical Outcome Study scale[16] might be useful, especially if followed by the Diagnostic Interview Schedule[17], screening version. The CES-D[18] or Zung[19] scales might be useful alternatives. None of these screening tests is available or used appropriately at this time. Treatment of depression is beneficial, although its availability is limited by toxic side-effects of medication and lack of resources for counseling and other psychological therapies.

Nearly 4% of the elderly suffer from unrecognized dementia or chronic cognitive impairment, although we recognize that in some populations the prevalence may be higher.[20] The prevalence of these conditions increases with age. The panel suggested that the Folstein Mini-Mental State[21] is the best screening procedure, although this is only useful in English-speaking populations. Although these tests are available, they are not currently used appropriately.

Currently no good treatment is available for 80% of elderly persons suffering from cognitive decline; the remaining 20% may be helped by treating reversible illness, including depression. The availability and benefit of treatment are highly dependent on the exact cause and could not be estimated overall.

Dermatological disorders

More than half of elderly persons have undetected dermatological disorders severe enough to lead to complications or complaints, with a higher incidence in sunny states such as California. The percentage of undetected disorders that do not result in the seeking of care could not be estimated. The most common diseases and conditions in this category are fungal infections, skin cancer, drug eruptions, photoaging, and xerosis. Visual inspection by a trained observer is the best screening procedure, although a history of symptoms of dry skin is also important for finding cases of xerosis.

TABLE 1—Characteristics of the Research Studies (N = 33)

Characteristic	No. of Documents	Reference Number[a]
Topics		
Hearing disorders	4	7, 6, 31, 32
Presbycusis		
Cerumen impaction		
Noise-induced loss		
Laboratory screening tests	4	1, 14, 15, 24
Diabetes		
Breast cancer		
Bacteriuria		
Hypercholesterolemia		
Anemia		
Colon cancer and bleeding colonic lesions		
Hypothyroidism		
Electrolyte disturbances		
Periodic medical review	0	—
Coronary artery disease		
Colon cancer		
Breast cancer		
Urinary incontinence		
Inappropriate use of medication		
Impaired social functioning		
Impaired physical functioning		
Nutritional problems		
Falls		
Hypertension	[b]	—
Ophthalmological disorders	0	—
Visual acuity		
Glaucoma		
Macular degeneration		
Cataracts		
Mental health	8	2, 8, 10, 11, 23, 28, 29, 33
Depression		
Dementia and cognitive impairment		
Dermatological disorders	4	3, 26, 27, 30
Skin cancer		
Xerosis		
Photoaging		
Fungal infections		
Drug eruptions		
Dental disorders	13	4, 5, 9, 12, 13, 16–22, 25
Xerostomia		
Caries		
Oral cancer		
Gingivitis and periodontitis		
Issues		
Effective screens	11	1, 3, 8, 10, 11, 14, 17, 23, 24, 30, 32
Subject characteristics	12	3–6, 11, 12, 19, 21, 25, 26, 28, 29
Rates of use	3	9, 18, 22
Incidence/prevalence	21	1, 3–5, 7, 13, 16–21, 25–27, 30
Supporting Agency		
Federal	16	3, 4, 9, 10, 12–14, 16, 18, 19–21, 29
Foundation	2	6, 22
University	3	26, 30, 31
Unidentified	6	7, 17, 23, 25, 27, 33
Starting date of data collection		
Before 1978	2	2, 13
1978–1979	4	1, 15, 23, 24
1980–1983	9	17–21, 25, 29
1984–1989	3	26, 27, 32
No exact dates given	15	3, 6–12, 14, 16, 22, 28, 30, 31, 33
Duration of study		
Under 1 year	1	23
Up to 1 year	6	1, 4, 18, 19, 21, 27
One to 2 years	3	15, 26, 32
2 or more years	6	2, 10, 14, 24, 25, 28

(Continued)

TABLE 1—Continued

Characteristic	No. of Documents	Reference Number[a]
Geographic location of sample		
National with Calif	2	2, 8
Regional without Calif	3	3, 11, 28
Calif statewide	1	27
One state (not Calif)	3	9, 17, 29
Local	16	4–7, 12, 15–22, 26, 30–33
Other	2	9, 17
Sampling		
Random	7	4, 5, 19–21, 27, 28
Population	2	1, 18
Data collection methods		
Record review	8	2, 7, 12, 14, 21, 24, 25, 27
Patient interview/ questionnaire	14	3, 5, 6, 8–11, 15, 17, 18, 22, 28, 29, 33
Provider survey	2	12, 33
Physical exam	14	1, 4, 5, 13, 16, 17, 20, 21, 23, 26, 30–32
Analysis by age		
	17	2–5, 12, 16–22, 25, 28–31
Study site		
Community	24	1, 3–6, 11–13, 15–22, 25–30, 32, 33
Chronic care institution	5	7, 14, 23, 24, 31
Age		
Includes elderly	2	1, 11
Elderly emphasized	11	1, 3, 7, 10, 12, 13, 17, 25, 26, 28–30
Exclusively elderly	17	4, 5, 6, 9, 14–16, 18–22, 24, 27, 31–33
Population/Ethnic composition		
White	23	2–8, 10–12, 16–23, 27, 29–31, 33
Hispanic	6	2, 3, 8, 10, 11, 27
Black	9	2, 3, 6, 8, 10, 11, 22, 27, 29
Asian	4	2, 8, 22, 27
Population/Income		
Middle	2	12, 15
Lower	3	9, 22, 24
Mixed	12	2, 6–8, 10, 14, 17, 28–32
Not stated	16	1, 3–5, 11, 13, 16, 18–21, 23, 25–27, 33

[a] Reference numbers refer to bibliography listing in Appendix B, not the the general reference list.

[b] Only review articles were included. See text for references.

Treatments for fungal infections, skin cancer, drug eruptions, and xerosis are both available and beneficial. The treatment of photoaging is probably not beneficial and generally not available, due to the cost of the medications and side-effects such as excessive skin dryness and pruritus.

Dental disorders

Nearly 50% of the elderly have undetected dental disease. The prevalence of undetected disease is likely to be highest for those with impaired mobility or limited finances. The most common diseases and conditions are caries, advanced gingivitis and periodontitis, xerostomia, and oral lesions including oral cancer. Clinical examination is the best screening procedure in all cases.

Beneficial treatments exist for caries, and, with certain limitations, for periodontitis, xerostomia, and oral lesions. The benefit of treatment of oral lesions depends on its characteristics. Treatment is available for each condition, although the availability of treatment for xerostomia is somewhat limited.

Literature Review. We reviewed 139 studies, 33 of which met our criteria for abstraction. We reviewed five pertaining to ophthalmology and three on periodic review of medical care but did not abstract data from them because they failed to meet our criteria. For most topics, the literature contributed limited data or no data at all.

Table 1 is a complete list of topics studied and the articles abstracted. Our review of the published literature disclosed a paucity of data on the effectiveness of screening. About two thirds of studies focused on the incidence and prevalence of disease, and the remainder emphasized the effectiveness of screening tests. Half the studies were supported by federal sources and one quarter used data collected between 1980 and 1983. In the 16 (48%) studies that specified duration, ten (33%) collected data for less than 2 years.

Most studies relied on local samples rather than statewide or national groups. Seven assessed randomly chosen samples, but none of these was a randomized controlled trial. Approximately 40% used physical examinations as the main source of data. More than half analyzed findings in terms of the age of the sample, and about half focused exclusively on the elderly. In 22 (66%) studies, participants were White.

Ten important review articles were identified that had examined the subject of screening for hypertension in the elderly.[22–31] Hypertension has been established as a risk factor for many serious problems, such as stroke, coronary artery disease, congestive heart failure, and multi-infarct dementia. Although some controversy still remains as to whether treating isolated systolic hypertension decreases mortality and morbidity, the general consensus is that treating both systolic and diastolic hypertension are useful and that detecting asymptomatic hypertension in the elderly is advantageous.

US Preventive Services Task Force. The Task Force provided data on 13 of this study's 34 specific topics. In some cases, the Task Force addressed issues that affect the elderly, but it did not specifically address whether screening needs were different in elderly populations. For four topics (hearing impairment, bacteriuria, elevated cholesterol, and visual acuity), the Task Force did specifically address the elderly, and in another three topics (glaucoma, dementia, and oral cancer), it reviewed topics that affect the elderly almost exclusively, although it did not specifically comment on the elderly. The Task Force recommended screening for hearing loss, elevated cholesterol, and loss of visual acuity.

Recommendations

Table 2 describes this study's recommendations and the bases on which they were made. We recommend the following without reservation: vision testing for refractive error; inspection of the skin surface for fungal infection, skin cancer, drug eruptions, and xerosis; interview for history of xerosis symptoms; audiometric testing for presbycusis and survey scales for hearing loss; otoscopic inspection for cerumen impaction; dental examination for caries; measurement of blood pressure

TABLE 2—Recommendations

Topic	Screen	PR	LT	PN	RXB	RXA	SC	TF	Recommendation[a]
Hearing loss									
Presbycusis	Audiometry	+	++	++	++	<>	<>	+	7
	Self-assessment scale	+	<>	+	++	<>	+	+	7
Cerumen impaction	Otoscopic inspection	++	<>	++	++	+	<>	<>	7
Noise-induced	Audiometry	<>	+	+	+	<>	<>	+	6
Screening laboratory tests									
Diabetes	Fasting blood sugar	+	–	+	+	++	<>	<>	6
Breast cancer	(see Periodic medical review)								
Bacteriuria	Urine analysis	++	–	+	<>	++	+	<>	6
Elevated cholesterol	Serum cholesterol	<>	–	+	<>	<>	–	+	5
Anemia	Hemoglobin or hematocrit	<>	–	+	<>	++	–	–	5
Bleeding colonic lesions	(see Periodic medical review)								
Hypothyroidism	TSH	<>	<>	+	++	++	<>	<>	6
Iatrogenic electrolyte disturbances	Serum electrolytes	<>	–	+	+	<>	<>	<>	5
Periodic medical review									
Coronary artery disease	History	<>	<>	+	+	+	<>	<>	6
Colon cancer	Guaiacs, sigmoidoscopy	<>	+	+	+	+	<>	<>	6
		<>	+	<>	+	+	<>	<>	6
Breast cancer	Breast exam, mammogram	+	+	+	+	+	<>	++	8
		+	+	+	+	++	<>	+	8
Urinary incontinence	History	+	<>	+	<>	<>	<>	<>	5
Inappropriate use of medications	Medication review	+	<>	+	++	<>	<>	<>	6
Social dysfunction	Assessment scales	+	<>	+	<>	–	+	<>	6
Physical dysfunction	Assessment scales	<>	<>	+	<>	<>	<>	<>	6
Nutrition	Height and weight	<>	<>	+	+	–	<>	<>	5
Falls	History (including safety)	<>	<>	+	<>	<>	<>	<>	5
Hypertension	Sphygmomanometer	+	++	++	+	+	+	+	8
Ophthalmological disorders									
Glaucoma	Tonometry, perimetry, ophthalmoscopy	+	<>	+	+	+	<>	–	6
		+	+	<>	+	+	<>	–	6
		+	<>	–	+	+	<>	–	5
Refractive error	Vision testing	++	<>	++	++	+	–	+	7
Cataracts	Vision testing	+	<>	+	+	++	–	<>	6
Macular degeneration	Vision testing	<>	<>	–	–	<>	–	<>	4
Diabetic retinopathy	Ophthalmoscopy, history and	<>	<>	–	+	+	<>	<>	5
	referral	<>	<>	++	+	+	–	<>	6
Mental health									
Depression	Screening questions	+	<>	+	<>	<>	<>	<>	6
Dementia	Mental status questions	<>	<>	+	<>	<>	<>	<>	6
Dermatological disorders									
Skin cancer	Visual inspection	+	+	+	+	++	<>	+	8
Xerosis	Visual inspection and history	+	<>	+	++	++	<>	<>	7
Photo aging	Visual inspection	+	<>	+	<>	<>	<>	<>	6
Fungal infection	Visual inspection	<>	<>	+	++	++	<>	<>	7
Drug eruptions	Visual inspection	<>	<>	+	++	++	<>	<>	7
Dental disorders									
Xerostomia	Clinical exam	<>	<>	++	+	<>	–	<>	6
Caries	Clinical exam	++	++	++	++	<>	–	<>	7
Oral cancer	Clinical exam	<>	<>	++	+	<>	–	+	6
Gingivitis and periodontitis	Clinical exam	+	<>	++	+	<>	–	<>	6

Note: PR = disease prevalence; LT = literature review; PN = recommendations of panels; RXB = treatment benefit; RXA = treatment availability; SC = current availability of screening; TF = US Preventive Services Task Force. – = negative support; <> = equivocal support or no data; + = supportive data; ++ = strongly supportive data.

[a] 9 = Strongly recommended; 8 = Recommended; 7 = Recommended with reservation; 6 = Equivocal; 5 = Equivocal; 4 = Equivocal; 3 = Not recommended with reservation, 2 = Not recommended; 1 = Strongly not recommended. The prevalence of disease was estimated by panelists when data were not available. The benefits and availability of treatment and the current availability of treatment were rated by panelists.

for hypertension; and breast examination and mammography for cancer.

Discussion

The recommendations presented here are applicable to public health screening programs that aim to refer large numbers of community-dwelling elders and rely on nurses who often travel to deliver services. This study focused on screening for programs staffed by public health nurses, so some tests receiving low ratings might be appropriate for programs relying on physicians. The ophthalmoscope, for example, is an effective screening device for glaucoma when used by a trained ophthalmologist.

From the perspective of clinical geriatrics, many of the tests and procedures that are not supported by this study may still be appropriate when performed by physicians and other health professionals and used for purposes other than screening. Recording mental status, for example, may provide useful baseline data for determining changes in mental status. Also, asking about symptoms of depression, which are often missed by primary care practitioners,[32] is generallly considered important in primary care geriatric medicine. Measuring renal function with serum creatine may also result in useful baseline data, and following serum electrolytes can be useful in persons on medications, such as nonsteroidal anti-inflammatory drugs, when renal function is known to be impaired. Reviewing medication use is almost certainly important in geriatric care when performed by a primary care physician, although screening for inappropriate medication use by others cannot be supported by our research. Thus, recommendations made here for the exclusion of certain measures from screening programs should not be interpreted to mean that these measures should be excluded from good clinical geriatric care.

We limited our investigation of screening to eight broadly defined topics, and these do not encompass all the needs of the elderly. We did not explore screening for vaccination status, for example, or for review of gastrointestinal problems or joint disease. A complete screening package might require other procedures or questions not discussed here. Moreover, the information on which we based our recommendations may change at any time. New research or alterations in reimbursement policies for hearing aids or dental work, for example, would necessitate changes in our ratings. If changes in reimbursement policies were to make hearing aids available to more persons, our ratings for audiometry would move from recommended with reservation to recommended or strongly recommended.

Better data are needed to guide screening programs, and research is needed on such topics as the response of patients and physicians to referrals from screening programs and their outcomes. We need, for example, to determine whether depression screening scales can be used efficiently and accurately in a population of elderly persons, whether physicians are responsive to referrals about depression, and whether patients receive beneficial treatments sooner because of screening. Research is also needed on ways to get patients to use their medications correctly. If inappropriate use (including noncompliance with therapy, use of interacting drugs, or use of drugs that might impair function or cognition) is uncovered, what should be done to alter patients' and physicians' behavior? In fact, additional research on screening is needed for each of the topics explored in this study.

We urge cautious confidence in applying our recommendations. Despite their limitations, they represent the best possible efforts of some leading experts in geriatric health care to weigh scientific and experimental evidence. It is obvious from this investigation, however, that more information is sorely needed to strengthen the foundation on which health policy on screening in the elderly can be established. □

Acknowledgments

This project was supported by a contract from the California Department of Public Health.

The authors are most grateful to Ms. Rhoda Slagel, of the California Preventive Health for the Aged Program. We are also indebted to Ms. Nancy Marr for assistance in conducting the literature review, assembling the bibliography, and scheduling the panels and to Ms. Ana Shiwoku for assistance in preparation of the manuscript and organization of the project.

References

1. *Aging America: Trends and Projections.* US Senate Committee on Aging in conjunction with the American Association of Retired Persons, the Federal Council on the Aging and the US Administration on Aging: 1987–1988 Edition.
2. Dawson D, Hendershot G, Fulton J. Aging in the eighties: functional limitations of individuals age 65 and over. *Advance Data,* No. 133. National Center for Health Statistics, June 10, 1987.
3. *[illegible]ear 2000 Health Promotion/Disease Pre-[illegible]ion Objectives for the Nation.* US Dept [illegible]ealth and Human Services. November 1[illegible]. Draft.
4. Bayer R, Moreno JD. Health promotion: ethical and social dilemmas of government policy. *Health Aff.* 1986;5:72–85.
5. Simon S, Wilson LB, Hermalin J, et al. Aging and Prevention: New Approaches for Preventing Health and Mental Problems in Older Adults. New York, NY: The Haworth Press; 1983.
6. Currie CT, Moore JT, Friedman SW, et al. Assessment of elderly patients at home: a report of fifty cases. *J Am Geriatr Soc.* 1981;29:398–401.
7. Rubenstein LZ, Josephson KR, Nicho-Seamons M, et al. Comprehensive health screening of well elderly adults: an analysis of a community program. *Gerontology.* 1986;41:342–352.
8. US Preventive Services Task Force. *Guide to Clinical Preventive Services.* Baltimore, Md: Williams & Wilkins; 1989.
9. Lawrence RS, Micralide AD, Kamero DB, Woolf SH. Report of the US Preventive Service Task Force. *JAMA.* 1990;263:436–437.
10. Park E, Fink A, Brook RH. Physicians ratings of appropriate indications for three procedures: theoretical indications vs. indications used in practice. *Am J Public Health.* 1989;79:445–447.
11. Park E, Fink A, Brook RH. Physicians ratings of appropriate indications for six medical-surgical procedures. *Am J Public Health.* 1986;76:766–772.
12. Canadian Task Force on Periodic Health Examination. *Can Med Assoc J.* 1979;121:1193–1254.
13. Fink A, Brook RH, Kosecoff J. Sufficiency of clinical literature on new appropriate uses of six medical and surgical procedures. *West J Med.* 1987;147:609–614.
14. Institute of Medicine. *Assessing Medical Technologies.* Washington, DC: National Academy Press, 1985. 258.
15. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 3rd ed, rev. Washington DC: American Psychiatric Association; 1987.
16. Burnam MA, Wells KB, Leake B, et al. Development of a brief screening instrument for detecting depressive disorders. *Med Care.* 1988;26:775–789.
17. Robins LN, Helzer JE, Crougham J, et al. National Institute of Mental Health Diagnostic Interview Schedule: its history, characteristics, and validity. *Arch Gen Psychiatry.* 1981;38:381–389.
18. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychiatr Meas.* 1977;1:385–401.
19. Zung WW. A self rating depression scale. *Arch Gen Psychiatry.* 1965;12:63–70.
20. Evans DA, Funkenstein HH, Albert MS, et al. Prevalence of Alzheimer's disease in a community population of older persons: higher than previously reported. *JAMA.* 1989;262:2551–2556.
21. Folstein MF, Folstein S, McHugh PR. Mini-Mental State: a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res.* 1975;12: 189–198.
22. Kannel WB. Prevalence, incidence, and hazards of hypertension in the elderly. *Am Heart J.* 1986;112:1362–1363.
23. Tuck ML, Griffiths RF, Johnson LE, et al. UCLA geriatric grand rounds: hypertension in the elderly. *J Am Geriatr Soc.* 1988;36:630–643.
24. Applegate WB, Dismuke SE, Runyan JW. Treatment of hypertension in the elderly: a time for caution? *J Am Geriatr Soc.* 1984;32:21–23.
25. Radin AM, Black HR. Hypertension in the elderly: the time has come to treat. *J Am Geriatr Soc.* 1981;29:193–200.
26. Gifford RW. Myths about hypertension in the elderly. *Med Clin North Am.* 1987;71:1003–1011.
27. O'Malley K, O'Callaghan W, O'Brien ET. Hypertension in the elderly: an overview. *Curr Med Res Opin.* 1982(7,suppl 1):53–62.
28. Smith WM. The case for treating hypertension in the elderly. *Am J Hypertens.* 1988;1:173S–178S.
29. Emeriau JP, Decamps A, Manciet G, et al. Hypertension in the elderly. *Am J Med.* 1978;82:1–8.
30. Applegate WB. Hypertension in elderly patients. *Ann Intern Med.* 1989;110:901–915.
31. Kannel WB, Dawber TR, McGee DL. Perspectives on systolic hypertension: the Framingham study. *Circulation.* 1980;61:1179–1182.
32. Wells KB, Stewart A, Hays RD, et al. The functioning and well-being of depressed patients: results from the medical outcomes study. *JAMA.* 1989;262:914–919.

APPENDIX A—References Reviewed but Not Abstracted

1. Albert M. Health screening to promote health for the elderly. *Nurse Pract.* 1987;12:42–58.
2. Arie T. Prevention of mental disorders of old age. *J Am Geriatr Soc.* 1984;32:460–465.
3. Beck JD, Lainson PA, Field HM, et al. Risk factors for various levels of periodontal disease and treatment needs in Iowa. *Community Dent Oral Epidemiol.* 1984;12:17–22.
4. Berwick DM. Screening for melanoma and skin cancer. *JAMA.* 1986;255:2443–2444.
5. Berwick DM. Screening in health fairs. *JAMA.* 1985;254:1492–1498.
6. Biro L, Price E. Skin cancer: screening in urban community. *NY State J Med.* 1978;78:753–755.
7. Blazer D. The epidemiology of late life depression. *J Am Geriatr Soc.* 1982;30:587–592.
8. Brody EM, Kleban MH. Physical and mental health symptoms of older people: who do they tell? *J Am Geriatr Soc.* 1981;29:442–449.
9. Brunelle JA, Miller-Chisholm AJ, Loe H. Oral health of United States adults: regional findings. Washington, DC: US Dept of Health and Human Services, publication NIH #88-2869;1988.
10. Burnam MA, Wells KB, Leake B, et al. Development of a brief screening instrument for detecting depressive disorders. *Med Care.* 1988;26:775–789.
11. Burns BJ, Burke JD. Improving health practices in primary care: findings from recent research. *Public Health Rep.* 1983;7:294–300.
12. Cebul RD, Beck JR. Biochemical profiles: applications in ambulatory screening and preadmission testing of adults. *Ann Intern Med.* 1987;106:403–413.
13. Chao A, Paganini-Hill A, Ross RK, et al. Use of preventive care by the elderly. *Prev Med.* 1987;16:710–722.
14. Cockburn J, Collin C. Measuring everyday memory in elderly people: a preliminary study. *Age Aging.* 1988;17:265–269.
15. Coetzee D. Secondary prevention of depressive illness among the elderly: a study of program effectiveness. *S Afr Med J.* 1980;58:571–574.
16. Coffey CE, Figiel GS, Djang WT, et al. Effects of ECT on brain structure: a pilot prospective magnetic resonance imaging study. *Am J Psychiatry.* 1988;145:701–706.
17. Coulehan JL, Zettler SM, Block M, et al. Recognition of alcoholism and substance abuse in primary care patients. *Arch Intern Med.*, 1987;147:349–352.
18. Davies JE, John DG, Jones AH, et al. Tympanometry as a screening test for treatable hearing loss in the elderly. *Br J Audiol.* 1988;22:119–121.
19. Davignon DD, Leshowitz BH. The speech-in-noise test: a new approach to the assessment of communications capability of elderly persons. *Int J Aging Hum Dev.* 1986;23:149–160.
20. Davis KL, Davis BM, Mathe AA, et al. Age and the dexamethasone suppression test in depression. *Am J Psychiatry.* 1984;141:872–874.
21. Douglass CW, Gillings D, Sollecito W, et al. National trends in the prevalence and severity of the periodontal diseases. *J Am Dent Assoc.* 1983;107:402-412.
22. Erkinjuntti T, Sulkava R, Wikstrom J, et al. Short portable mental status questionnaire as a screening test for dementia and delirium among the elderly. *J Am Geriatr Soc.* 1987;35:412–416.
23. Ewing JA. Detecting alcoholism: the CAGE questionnaire. *JAMA.* 1984; 252:1905–1907.
24. Falkenberg M, Kagedal B. Hyperthyroidism in elderly women: registered and detected by screening in a primary health care district. *Scand J Prim Health Care.* 1985;3:11–13.
25. Feldman RS, Alman J, Muench ME, et al. Longitudinal stability and masticatory function of human dentition. *Gerodontology.* 1984;3:107–113.
26. Field SI. Melanoma/skin cancer screening in Michigan. *J Am Acad Dermatol.* 1987;16:578–583.
27. Fleischer DE, Goldberg SB, Browning TH, et al. Detection and surveillance of colorectal cancer. *JAMA.* 1989;261:580–585.
28. Gallagher D. Assessing affect in the elderly. *Clin Geriatr Med.* 1987;3:65–85.
29. Gilchrest BA. Geriatric skin problems. *Hosp Pract.* 1986;21:55–65.
30. Gilchrest BA. Some gerontologic considerations in the practice of dermatology. *Arch Dermatol.* 1979;115:1343–1346.
31. Glass RL, Alman JE, Chauncey HH. A ten-year longitudinal study of caries incidence rates in a sample of male adults in the USA. *Caries Res.* 1987;21:360–367.
32. Gordon ST, Jahnigen DW. Oral assessment of the edentulous elderly patient. *J Am Geriatr Soc.* 1983;31:797–801.
33. Gron P. Preventive dental health program for the elderly: rationale and preliminary findings. *Spec Care Dent.* 1981;3:129–132.
34. Guidelines for screening for hearing impairment and middle ear disorders. *ASHA.* 1989;31:71–77.
35. Guidelines for identification of audiometry. *ASHA.* 1975;17:94–99.
36. Guidelines for acoustic immitance screening of middle-ear function. *ASHA.* 1979;21:283–288.
37. Guralnik JM, Harris TB, White LR, et al. Occurrence and predictors of pressure sores in the National Health and Nutrition Examination Survey follow-up. *J Am Geriatr Soc.* 1988;36:807–812.
38. Gurland B, Dean L, Cross P, et al. The epidemiology of depression and dementia in the elderly: the use of multiple indicators of these conditions. In: Cole J, Barrett J, eds. *Psychopathology of the Aged.* American Psychopathological Association Series. New York, NY: Raven Press; 1980.
39. Hand JS, Whitehill JM. The prevalence of oral mucosal lesions in an elderly population. *J Am Dent Assoc.* 1986;112:73–76.
40. Ismail AI, Burt BA, Hendershot GE, et al. Findings from the Dental Care Supplement of the National Health Interview Survey, 1983. *J Am Dent Assoc.* 1987;114:617–621.
41. Hand JS, Hunt RJ, Beck JD. Incidence of coronal and root caries in an older adult population. *J Public Health Dent.* 1988;48:14–19.
42. Hankin J. Management of emotionally distrubed patients in primary care settings: a review of the North American literature. Mental Health Services in Primary Care Settings/Health Services Reports. US Dept of HHS Series DN No. 2.
43. Haug K, Bakke A, Daae LN, et al. Screening for hematuria, glycosuria and proteinuria in people aged 55–64: technical, clinical, and cost-benefit experience from a pilot study. *Scand J Prim Health Care.* 1985;3:31–34.
44. Hayes D. *Aging and Speech Understanding. Seminars in Hearing.* New York, NY: Thieme-Stratton, Inc.; 1985.
45. Hitchings RA. Glaucoma screening in the elderly. *Curr Med Res Opin.* 1982;7(suppl. 1):23–27.
46. Hoeper EW, Nycz GR, Cleary PD, et al. Estimated prevalence of RDC mental disorder in primary medical care. *Int J Ment Health.* 1979;8:6–15.
47. Hyman L. Epidemiology of eye disease in the elderly. *Eye.* 1987;1:330–341.
48. Jorm AF, Korten AE. Assessment of cognitive decline in the elderly by informant interview. *Br J Psychiatry.* 1988;152:209–213.
49. Kamerow DB. Is screening for mental health problems worthwhile in family practice. *J Fam Pract.* 1987;25:181–183.
50. Kane RL, Kane RA, Arnold SB. Prevention and the elderly: risk factors. *Health Serv Res.* 1985;19:945–1006.
51. Keltner JL, Johnson CA. Visual function, driving safety, and the elderly. *Ophthalmology.* 1987;94:1180–1188.
52. Klerman GL, Budman S, Berwick D, et al. Efficacy of a brief psychosocial intervention for symptoms of stress and distress among patients in primary care. *Med Care.* 1987;25:1078–1088.
53. Knight KK, Fielding JE, Battista RA. Occult blood screening for colorectal cancer. *JAMA.* 1989;261:587–593.

(continued)

APPENDIX A—continued

54. Koenig HG, Meador KG, Cohen HJ, et al. Self-rated depression scales and screening for major depression in the older hospitalized patient with medical illness. *J Am Geriatr Soc.* 1988;36:699–706.
55. Larson EB, Reifler BV, Sumi SM, et al. Diagnostic tests in the evaluation of dementia: a prospective study of 200 elderly outpatients. *Arch Intern Med.* 1986;146:1917–1922.
56. Lebel JO. Health needs of the elderly. *Dent Clin North Am.* 1989;33:1–5.
57. Lichtenstein MJ, Bess FH, Logan SA. Validation of screening tools for identifying hearing-impaired elderly in primary care. *JAMA.* 1988;259:2875–2878.
58. Lieffel ED, Watson W. Screening for skin cancer at a country fair. *West J Med.* 1975;122:123–126.
59. Linn LS, Yager J. The effect of screening, sensitization, and feedback on notation of depression. *J Med Educ.* 1980;55:942–949.
60. Lynch SR, Finch CA, Monsen ER, et al. Iron status of elderly Americans. *Am J Clin Nutr.* 1982;36:S1032–S1045.
61. Mader S. Hearing impairment in elderly persons. *J Am Geriatr Soc.* 1984;32:548–553.
62. Magni G, Schifano F, DeLeo D. Assessment of depression in an elderly medical population. *J Affective Disord.* 1986;11:121–124.
63. Maize JC, Foster G. Age-related changes in melanocytic naevi. *Clin Exp Dermatol.* 1979;4:49–58.
64. Mandel ID. Preventive dental services for the elderly. *Dent Clin North Am.* 1989;33:81–90.
65. Marshall L. Audiological assessment of older adults. *Semin Hear.* 1985;6:162–180.
66. Mohart RE, Davis ME, Weiss DG, et al. Dental health status of an aging VA population: implications for a preventive dental health care program. *Health Serv Res.* 1986;10:933–946.
67. Moore JT, Silimperi DR, Bobula JA. Recognition of depression by family medicine residents: the impact of screening. *J Fam Pract.* 1978;7:509–513.
68. Morgan PP. Canadian task force on the periodic health examination. *Can Med Asoc J.* 1979;121:1–45.
69. Niessen LC, Jones JA. Oral health changes in the elderly. *Postgrad Med.* 1984;75:231–237.
70. Oboler SK, LaForce FM. The periodic physical examination in asymptomatic adults. *Ann Intern Med.* 1989;33:325–329.
71. Orentreich N, Markosfky J, Vogelman JH. The effect of aging on the rate of linear nail growth. *J Invest Dermatol.* 1979;73:126–130.
72. Ow CL, Leman HJ, Weaver MJ. Does screening proctosigmoidoscopy result in reduced mortality from colorectal cancer? *J Gen Intern Med.* 1989;4:209–215.
73. Parmelee PA, Katz IR, Lawton MP. Depression among institutionalized aged: assessment and prevalence estimation. *Gerontology.* 1989;44:M22–M29.
74. Plewig G, Kligman AM. Proliferative activity of the sebaceous glands of the aged. *J Invest Dermatol.* 1978;70:314–317.
75. Pochi PE, Strauss JS, Downing DT. Age-related changes in sebaceous gland activity. *J Invest Dermatol.* 1979;73:108–111.
76. Potts RO, Buras EM, Chrisman DA. Changes with age in the moisture content of human skin. *J Invest Dermatol.* 1984;82:97–100.
77. Prevalence of dermatological disease among persons 1–74 years of age: United States. *Advance Data*, No. 4. National Center for Health Statistics. 1977.
78. Randolph LJ, Schow RL. Threshold inaccuracies in an elderly clinical population: ear canal collapse as a possible cause. *J Speech Hear Res.* 1983;26:54–58.
79. Resnick SB. Hearing. *Md Med J.* 1989;38:138–139.
80. Rigel DS, Friedman RJ, Kopf AW, et al. Importance of complete cutaneous examination for the detection of malignant melanoma. *J Am Acad Dermatol.* 1986;14:857–860.
81. Ritchie K. The screening of cognitive impairment in the elderly: a critical review of current methods. *J Clin Epidemiol.* 1988;41:635–643.
82. Roberts RE, Vernon SW. Depression in the community. *Arch Gen Psychiatry.* 1982;39:1407–1409.
83. Robins LN, Helzer JE, Weissman MM, et al. Lifetime prevalence of specific psychiatric disorders in three sites. *Arch Gen Psychiatry.* 1984;41:949–958.
84. Rogers J, Grower R, Supino P. Target groups for screening elderly outpatients. *Am J Prev Med.* 1988;4:27–34.
85. Sanderson D. Ocular screening for the elderly. *Can Nurse.* 1986;82:19–20.
86. Schow RL, Nerbonne MA. Communication screening profile: use with elderly clients. *Ear Hear.* 1982;3:135–147.
87. Scotto J, Kopf AW, Urbach F. Non-melanoma skin cancer among Caucasians in four areas of the United States. *Cancer.* 1974;34:1333–1338.
88. Somers AR, Kleinman L, Clark WD. Preventive health services for the elderly: the Rutgers Medical School project. *Inquiry.* 1982;19:190–198.
89. Sommer A. Eye care for the elderly: looking better! seeing better! *Ophthalmology.* 1987;94:1178–1179.
90. Strauss RP, Hairfield WM, George MC. Disabled adults in sheltered employment: an assessment of dental needs and costs. *Am J Public Health.* 1985;75:661–663.
91. Sweer L, Martin DC, Ladd RA, et al. The medical evaluation of elderly patients with major depression. *Gerontology.* 1988;43:M53–M58.
92. Thomas PD, Hunt WC, Gary PJ. Hearing acuity in a healthy elderly population: effects on emotional, cognitive, and social status. *Gerontology.* 1983;38:321–325.
93. Thompson LW, Gallagher D, Breckenridge JS. Comparative effectiveness of psychotherapies for depressed elders. *J Consult Clin Psychol.* 1987;55:385–390.
94. Tulloch AJ, Moore V. A randomized controlled trial of geriatric screening and surveillance in general practice. *J R Coll Gen Pract.* 1979;29:733–742.
95. Ventry IM, Weinstein BE. The hearing handicap for the elderly: a new tool. *Ear Hear.* 1982;3:128–134.
96. Ventry IM, Weinstein BE. Identification of elderly people with hearing problems. *ASHA.* 1983;7:37–42.
97. Weary PE. A two-year experience with a series of rural skin and oral cancer detection clinics. *JAMA.* 1971;217:1862–1863.
98. Wehr TA, Sack DA, Rosenthal NE, et al. Rapid cycling affective disorder: contributing factors and treatment responses in 51 patients. *Am J Psychiatry.* 1988;145:179–184.
99. Weiler PG, Chi I, Lubben JE. A statewide preventive health care program for the aged. *Public Health Rep.* 1989;104:215–221.
100. Weiner RD. The role of electroconvulsive therapy in the treatment of depression in the elderly. *J Am Geriatr Soc.* 1982;30:710–712.
101. Weinstein BE, Ventry IM. Audiologic correlates of hearing handicap in the elderly. *J Speech Hear Res.* 1983;26:148–151.
102. Wells KB. *Depression as a Tracer Condition for the National Study of Medical Care Outcomes.* Rand Corporation, Santa Monica, CA, R-3293-RWJ. 1985.
103. Wolf-Klein GP, Holt T, Silverstone FA, et al. Efficacy of routine annual studies in the care of elderly patients. *J Am Geriatr Soc.* 1985;33:325–329.
104. Woo B, Woo B, Cook F, et al. Screening procedures in the asymptomatic adult. *JAMA.* 1985;254:1480–1499.
105. OTA questions costs and effectiveness of cholesterol screening in the elderly. *ASCP News* (Newsletter of the American Society of Clinical Pathologists). 1989.
106. Zung WK, Magill M, Moore JT, et al. Recognition and treatment of depression in a family medicine program. *J Clin Psychiatry.* 1983;44:3–6.

APPENDIX B—Abstracted Literature

1. Allison JE, Feldman R. Cost benefits of hemocult screening for colorectal carcinoma. *Dig Dis Sci.* 1985;30:860–865.
2. Balzer DG, Bachar JR, Manton KG. Suicide in late life: review and commentary. *J Am Geriatr Soc.* 1986;34:519–525.
3. Beauregard S, Gilchrest BA. A survey of skin problems and skin care regimens in the elderly. *Arch Dermatol.* 1987;123: 1638–1643.
4. Beck JD, Hunt RJ, Hand JS, et al. Prevalence of root and coronal caries in a noninstitutionalized older population. *J Am Dent Assoc.* 1985;111:964–967.
5. Beck JD, Kohout FJ, Heckert DA. Root caries: physical, medical and psychosocial correlates in an elderly population. *Gerodontics.* 1986;3:242–247.
6. Bess FH, Lichtenstein MJ, Logan SA. Hearing impairment as a determinant of function in the elderly. *J Am Geriatr Soc.* 1989;37:123–128.
7. Bingea RL, Raffin M, Aune KJ, et al. Incidence of hearing loss among geriatric nursing-home residents. *J Aud Res.* 1982;22;275–283.
8. Blazer DG, Hughes DC, George LK. The epidemiology of depression in an elderly community population. *Gerontologist.* 1987;27:281–287.
9. Branch LG, Antczak AA, Stason WB. Toward understanding the use of dental services by the elderly. *Spec Care Dent.* 1986;6:38–41.
10. Carvey AJ, Douglass CW, Chauncey HH. Personality factors, demographic variables, and indexes of periodontal disease in normal, community-dwelling males. *Spec Care Dent.* 1986;6:170–174.
11. Chauncey HH, Baric JM, Alman JE, et al. Longitudinal study of tooth loss and caries prevalence in healthy male adults. *Gerodontics.* 1987;3:38–42.
12. Domoto K, Ben R, Wei JY, et al. Yield of routine annual laboratory screening in the institutionalized elderly. *Am J Public Health.* 1985;75:243–245.
13. Gallagher DE, Thompson LW. Effectiveness of psychotherapy for both endogenous and nonendogenous depression in older adult outpatients. *Gerontology.* 1983;38:707–712.
14. Holzer CE, Tischler GL, Leaf PJ, et al. An epidemiologic assessment of cognitive impairment in a community population. *Res Comm Mental Health.* 1984;4:3–32.
15. Hale WE, Perkins LL, May FE, et al. Vitamin E effect on symptoms and laboratory values in the elderly. *J Am Diet Assoc.* 1986;86:625–629.
16. Hand JS, Hunt RJ. The need for restorations and extractions in a noninstitutionalized elderly population. *Gerodontics.* 1986;2:72–76.
17. Heine CS, Jong A, Casamassimo PS, et al. Oral health status and behaviors of the elderly and other adults: results of a health screening. *Spec Care Dent.* 1983;5:217–221.
18. Hunt RJ. Periodontal treatment needs in an elderly population in Iowa. *Gerodontics.* 1986;2:24–27.
19. Hunt RJ, Srisilapanan P, Beck JD. Denture related problems and prosthodontic treatment needs in the elderly. *Gerodontics.* 1985;1:226–230.
20. Hunt RJ, Beck JD, Lemke JH, et al. Edentulism and oral health problems among elderly rural Iowans: the Iowa 65+ rural health study. *Am J Public Health.* 1985;75:1177–1181.
21. Hunt RJ, Field HM, Beck JD. The prevalence of periodontal conditions in a noninstitutionalized elderly population. *Gerondontics.* 1985;1:176–180.
22. Kiyak HA. An explanatory model of older persons' use of dental services: implications for health policy. *Med Care.* 1987;25:936–952.
23. Kolman P, Phil M. The value of laboratory investigations of elderly psychiatry patients. *J Clin Psychiatry.* 1984;45:112–116.
24. Levinstein MR, Ouslander JG, Rubenstein LZ, et al. Yield of routine annual laboratory tests in a skilled nursing home population. *JAMA.* 1987;258:1909–1915.
25. Linden G. Periodontal destruction and loss of the remaining natural teeth. *Community Dent Oral Epidemiol.* 1988;16:19–21.
26. Lookingbill DP, Hershey PA. Yield from a complete skin examination: findings in 1157 new dermatology patients. *J Am Acad Dermatol.* 1988;18:31–37.
27. Luckmann R, Weiler PG. Screening the elderly for skin disease: A report of the experience of the California Preventive Health Care for the Aging Program. *Fam Community Health.* 1988;11:53–64.
28. Meyers JK, Weissman MM, Tischler GL, et al. Six-month prevalence of psychiatric disorders in three communities. *Arch Gen Psychiatry.* 1984;41:959–967.
29. Murrell SA, Himmelfarb S, Wright K. Prevalence of depression and its correlates in older adults. *Am J Epidemiol.* 1983;117:173–185.
30. Olsen T, Feeser TA, Conte ET, et al. Skin cancer screening: a local experience. *J Am Acad Dermatol.* 1987;16:637–641.
31. Schow W, Nerbonne MA. Hearing levels among elderly nursing home residents. *J Speech Hear Disord.* 1989;45:124–132.
32. Uhlmann RF, Larson EB, Rees TS. Relationship of hearing impairment to dementia and cognitive dysfunction in older adults. *JAMA.* 1989;261:1916–1919.
33. Waxman HM, Carner EA. Physicians' recognition, diagnosis, and treatment of mental disorders in elderly medical patients. *Gerontologist.* 1984;24:593–597.

The Increasing Supply of Physicians in US Urban and Rural Areas, 1975 to 1988

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ABSTRACT

Background. Despite the rapid growth of the US physician supply since the mid-1970s, it remains unclear whether physicians have spread into the most rural areas of the country. This report examines the urban–rural distribution of physicians between 1975 and 1988.

Methods. A county-based typology of the urban–rural continuum was employed to examine trends in the supply of nonfederal primary care physicians, specialist physicians, and osteopaths.

Results. All urban and rural areas gained physicians during the late 1970s and 1980s. The supply of physicians increased most rapidly in metropolitan counties. Within nonmetropolitan areas, urbanized remote counties became more prominent centers of the physician supply. Osteopaths were more likely to locate in the most rural areas than allopaths. The physician supply in all areas also became more specialized over time.

Conclusions. The rapid growth of the US physician supply was associated with the spread of more practitioners into all parts of the country. However, the supply of physicians increased most rapidly in urban areas, widening urban–rural differences in the availability of physicians. (*Am J Public Health.* 1991; 81:1141–1147)

Introduction

The number of nonfederal allopathic and osteopathic physicians in the United States increased nearly four times more rapidly than the population between 1975 and 1988. Despite the rapid increase, physicians remained concentrated in urban centers. By 1988 the ratio of physicians per 100 000 persons was 226 in metropolitan areas and only 98 in nonmetropolitan areas. This gap has persisted despite government efforts to place more physicians in rural communities and concerns about an imminent national surplus of physicians.

The uneven distribution of physicians is partly due to the concentration of medical education, research, and specialized care in urban central places. However, there is much evidence that physicians prefer urban locations for other reasons, including the personal ties developed during medical training, the greater opportunities to interact with professional colleagues, and a taste for urban amenities.[1] The disproportionate tendency of physicians to locate in urban areas narrows the choice of practitioners for rural populations and suggests that physician services are relatively less accessible in rural areas.

The federal government first began to address the geographic maldistribution of physicians during the early 1970s, when a variety of measures were enacted to increase the supply of practitioners in inner cities and rural areas.[2,3] One of the most important measures was the National Health Service Corps (NHSC) scholarship program, which provided financial aid to medical students and other health trainees in return for service after graduation in designated Health Personnel Shortage Areas (HPSAs, formerly termed Health Manpower Shortage Areas).

Federal efforts to increase the number of physicians in underserved areas were curtailed during the early 1980s, and the NHSC scholarship program was gradually phased out.[2,4] The shift in policy was influenced by the 1980 report by the Graduate Medical Education National Advisory Committee (GMENAC), which predicted that a substantial surplus of physicians would emerge by 1990.[5] Federal officials also contended that market forces alone would be sufficient to attract more physicians to rural areas as competition intensified among physicians in urban centers.[2]

A series of studies by the RAND Corporation during the early 1980s provided evidence that physicians' location decisions had been affected by market forces.[6–10] The studies showed that the proportion of small towns served by various kinds of specialists had increased during the 1970s until nearly every urban place with 2500 or more inhabitants had at least one physician. The RAND researchers argued that physicians had responded to the increasing physician supply in accordance with economic location theory by diffusing to smaller places until all towns above a critical size had at least one physician. They also predicted that physicians would spread more widely in rural

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This paper was submitted to the journal December 26, 1990, and accepted with revisions April 23, 1991.

areas during the 1980s as the growth of the physician population continued.[7]

In contrast, trends in physician/population ratios during the 1970s and early 1980s led other observers to conclude that physicians had not spread into rural areas in substantial numbers.[11–14] Metropolitan (metro) counties continued to have much higher physician/population ratios than nonmetropolitan (nonmetro) counties. Thus, physicians remained concentrated in urban centers. Further classification of counties by population size also showed that physican/population ratios had increased most slowly in small nonmetro counties. This trend was interpreted as evidence that few physicians had moved into the most rural areas of the country.[11,13]

Continued public concern about physician shortages led to new federal initiatives to place physicians in underserved communities during the late 1980s, including the NHSC loan repayment program and a Medicare reimbursement bonus for services performed by physicians in designated HPSAs.[4] Funding for the NHSC scholarship program was also significantly increased starting in 1991.[15] The new initiatives reveal that some areas remained unattractive to physicians despite the rapid growth of the physician supply.

The purpose of this report is to examine the growth of the nonfederal physician supply since the mid-1970s during a period of major changes in federal health personnel policy. A county-based typology of the urban–rural continuum will be employed to determine whether the supply of physicians increased in predominantly rural areas without sizeable towns, as well as in urbanized rural areas and smaller and larger metro centers. These distinctions are important because the RAND studies demonstrated that town size plays a critical role in physicians' location decisions. Other studies of the physician supply based on the classification of counties by population size have failed to identify predominantly rural areas because nonmetro counties with small populations may lie near metro centers or contain relatively large urban places.

Methods

Information about the annual distribution of patient care allopaths (MD physicians) and active osteopaths (DO physicians) by county was obtained from the 1989 Area Resource File and the 1975 to 1988 MD/DO Enhanced County File prepared by the Office of Data Analysis and Management, US Department of Health and Human Services. The data for allopaths and osteopaths were not always available for the same year. Federal physicians were excluded because most served special populations. Alaska was also excluded because the physician data for the state were not broken down by county. Additional information about the distribution of the resident population by county was obtained from the US Bureau of the Census intercensal population estimates. Counties affected by border changes between 1975 and 1988 were combined into larger units with constant boundaries.

Nonfederal allopaths were divided into two groups according to self-designated practice specialty: (1) primary care physicians providing patient care in general/family practice, internal medicine, obstetrics/gynecology, or pediatrics; and (2) specialist physicians providing patient care in any other specialty. Nonfederal active osteopaths were combined in a third category. Resident physicians were included in the appropriate category because they provided a substantial amount of patient care. Internal medicine subspecialist physicians were placed in the primary care category because they could not be distinguished from general internists prior to 1986. The small proportion of physicians who did not report a specialty were excluded from the analysis. Active osteopaths included both patient care and non–patient care physicians because the two groups could not be distinguished after 1981, when nearly 95% were engaged in patient care.

All counties were classified into 10 urban–rural continuum categories by using a typology developed by the Economic Research Service, US Department of Agriculture.[16] Counties were first divided into metro and nonmetro areas by using the 1983 US Office of Management and Budget definition of Metropolitan Statistical Areas (MSAs). Metro counties were then subdivided by 1980 MSA population and structure, and nonmetro counties were subdivided by 1980 county urban population and proximity to MSAs (Table 1). Variations in the level of urban influence are thus measured by the population and structure of MSAs in metro areas and by county urban populations and proximity to MSAs in nonmetro areas. Counties toward the urban end of the continuum are described as "more urban" and those toward the rural end are described as "more rural" for convenience.

The supply of physicians in each category was measured by the ratio of physicians per 100 000 residents. Separate ratios were calculated for primary care and specialist physicians for 1975, 1981, and 1988, and for osteopaths for 1976, 1981, and 1987. Since the reporting date for physicians shifted from December 31 to January 1 in 1988, the 1988 ratios were based on the 1987 resident population to increase comparability over time. The 1981 to 1988 period was only 1 day longer than the 1975 to 1981 period because of the shift in dates.

Changes in the physician supply over time were measured by calculating the mean annual change in the physician/population ratios for each urban–rural continuum category. This measure of absolute change was chosen to compare areas with different initial ratios.

Results

Primary Care Physicians

The total number of primary care physicians grew 3.8% per year between 1975 and 1988, rising from 132 000 to 207 700. During the same period the overall ratio of primary care physicians per 100 000 persons increased from 61.4 to 85.5 (Table 2). However, the ratio was substantially higher at the more urban end of the urban–rural continuum than at the more rural end (Figure 1). The central areas of large MSAs (large metro core counties) had the highest ratio throughout the period. The suburbs of large MSAs (large metro fringe counties) and medium metro counties had the next highest ratios. Small metro counties had lower ratios than other metro counties, but higher ratios than nonmetro counties.

Within nonmetro areas, urbanized counties had higher primary care ratios than less urbanized or rural counties. However, within each urban population category, remote counties some distance from MSAs had more primary care physicians than adjacent counties near MSAs. This pattern indicates that nonmetro primary care physicians preferred to locate in areas where distance made metro physician services less accessible to local residents.

The mean annual change in the overall primary care physician ratio was nearly constant, varying from 1.98 in 1975 to 1981 to 2.04 in 1981 to 1988 (Table 2). On average, communities thus gained about two primary care physicians per 100 000 residents per year during the late 1970s and 1980s. Although all urban and rural areas gained primary care physicians, the annual gains were greatest in metro counties and nonmetro urbanized remote counties. All other nonmetro counties added primary care physicians much more slowly.

The faster growth of the primary care physician supply in more urban areas caused urban–rural differences in the availability of practitioners to widen over time. The gap between metro and nonmetro areas expanded from 30 to 45 primary care physicians per 100 000 persons between 1975 and 1988 (Table 2). Differences within nonmetro areas also increased as nonmetro urbanized remote counties (and to a lesser extent, less urbanized remote counties) became more prominent centers of the primary care physician supply (Figure 1). The emergence of these centers indicates that nonmetro primary care physicians have become increasingly concentrated in counties with larger urban places, even though all areas gained practitioners.

Nonmetro counties gained primary care physicians more rapidly during the 1980s than the late 1970s (Table 2). In contrast, most metro counties (except large metro fringe counties) gained primary care physicians more slowly. The shift in growth rates indicates that nonmetro areas and the suburbs of large MSAs became more attractive to primary care physicians after 1981.

The urban–rural differences in the supply of primary care physicians were affected by the classification of internal medicine subspecialists as primary care physicians. However, subspecialists accounted for only a small part of the differences in 1988 when subspecialists could be distinguished from general internists. In that year 8% of the primary care physicians in metro areas and 3% of those in nonmetro areas were actually subspecialists.

In contrast, resident physicians accounted for a larger share of the urban–rural differences in the primary care physician supply. More than 20% of the primary care physicians in metro areas were in residency training in 1988, in comparison with only 4% of those in nonmetro areas. There was little change in these proportions over time. The differences in the availability of primary care physicians would be smaller if residents were excluded, but the important role of residents in staffing urban teaching hospitals would be ignored.

Specialist Physicians

During 1975 to 1988 the number of specialist physicians grew more rapidly than the number of primary care physi-

TABLE 1—County Urban–Rural Continuum Categories

Urban–Rural Continuum Category	Number of Counties	Percent of 1987 Resident Population
Total counties[a]	3070	100.0
Metro counties	710	76.8
1. Large metro counties		
a. Core counties containing primary central city of MSAs with 1980 populations of 1 000 000 or more	55	29.0
b. Fringe (suburban) counties of MSAs with 1980 populations of 1 000 000 or more	169	16.3
2. Medium metro counties in MSAs with 1980 populations of 250 000 to 999 999	289	22.4
3. Small metro counties in MSAs with 1980 populations under 250 000	197	9.1
Nonmetro Counties	2360	23.2
1. Urbanized counties with 20 000 or more urban residents in 1980		
a. Adjacent to an MSA[b]	137	4.4
b. Remote (not adjacent to an MSA)	150	3.7
2. Less urbanized counties with 2500 to 19 999 urban residents in 1980		
a. Adjacent to an MSA[b]	551	6.2
b. Remote (not adjacent to an MSA)	748	6.3
3. Rural counties with fewer than 2500 urban residents in 1980		
a. Adjacent to an MSA[b]	228	1.1
b. Remote (not adjacent to an MSA)	546	1.6

[a] Excluding Alaska.

[b] Counties were considered adjacent to an MSA when the county and MSA borders touched at more than one point and 2% or more of the county labor force commuted to the central county of the MSA.

Note: Metro = metropolitan; MSA = Metropolitan Statistical Area.

TABLE 2—Nonfederal Primary Care Physicians per 100 000 Resident Population by Urban–Rural Continuum Category, 1975–1988[a]

Urban–Rural Continuum Category	Primary Care Physician/ Population Ratio			Mean Annual Change in Primary Care Physician/ Population Ratio	
	1975	1981	1988	1975–1981	1981–1988
US Total	61.4	73.3	85.5	1.98	2.04
Metro	68.5	82.3	95.9	2.31	2.26
Large metro					
Core	86.6	101.9	116.0	2.56	2.34
Fringe	55.9	69.6	85.7	2.28	2.68
Medium metro	59.3	72.9	85.1	2.28	2.02
Small metro	52.0	65.1	76.2	2.18	1.86
Nonmetro	38.6	44.4	51.3	0.97	1.14
Urbanized					
Adjacent to MSA	42.1	47.9	54.8	0.98	1.14
Remote	46.3	57.8	69.3	1.91	1.92
Less urbanized					
Adjacent to MSA	35.4	39.1	44.2	0.61	0.86
Remote	39.1	45.0	51.8	0.98	1.13
Rural					
Adjacent to MSA	28.1	29.0	31.9	0.16	0.48
Remote	29.0	32.8	38.2	0.63	0.91

[a] Excluding Alaska.

Note: Metro = metropolitan; MSA = Metropolitan Statistical Area.

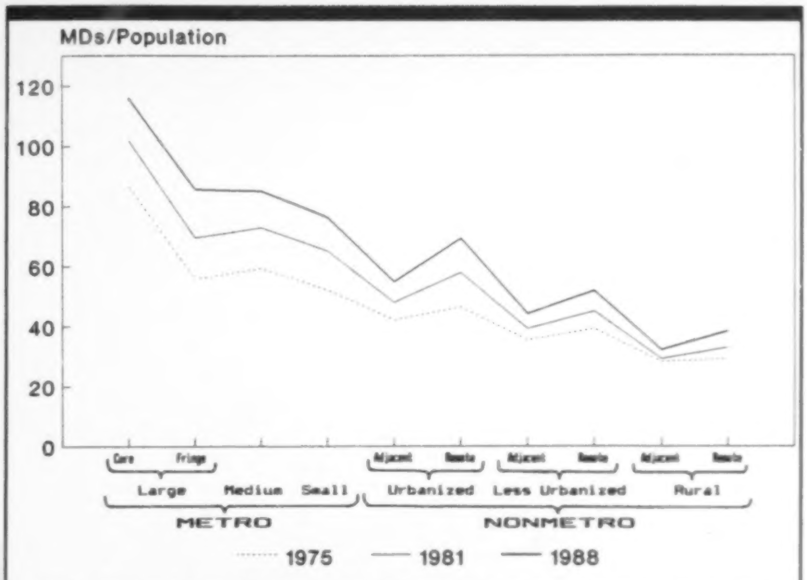

FIGURE 1—Nonfederal Primary Care Physicians per 100 000 persons by Urban–Rural Continuum Category, 1975–1988

cians, increasing by 4.2% per year from 147 200 to 242 900. As a result, the overall specialist physician population ratio rose from 68.4 to 100.0 (Table 3). The urban–rural distributions of specialists and primary care physicians were very similar (Figure 2). However, there were even greater differences between more urban and more rural areas in the supply of specialists. The specialist ratio was highest in the central areas of large MSAs and lowest in nonmetro rural counties. Within nonmetro areas, remote counties had more specialists than adjacent counties in the same urban population category.

The total supply of specialists grew more rapidly during the 1980s than the late 1970s (Table 3). All urban and rural areas gained specialists, but the gains were greater in more urban areas, repeating the pattern among primary care physicians. The increase in the specialist supply was particularly rapid in large MSAs, which gained more than three specialists per 100 000 persons per year. In contrast, the specialist supply grew only one tenth as rapidly in nonmetro rural counties.

Urban–rural differences in the availability of specialists widened over time due to the faster growth of the specialist ratio in more urban areas (Table 3). Between 1975 and 1988 the gap between metro and nonmetro areas expanded from 56 to 78 specialists per 100 000 persons. In nonmetro areas, urbanized remote counties also became more prominent centers of the specialist supply (Figure 2). Most nonmetro areas (except rural adjacent counties) gained specialists more slowly after 1981, but the central areas of large MSAs began gaining specialists even more rapidly.

TABLE 3—Nonfederal Specialist Physicians per 100 000 Resident Population by Urban–Rural Continuum Category, 1975–1988*

Urban–Rural Continuum Category	Specialist Physician/ Population Ratio 1975	1981	1988	Mean Annual Change in Specialist Physician/ Population Ratio 1975–1981	1981–1988
US Total	68.4	83.6	100.0	2.53	2.74
Metro	81.7	99.3	118.2	2.94	3.14
Large metro					
Core	105.1	124.3	145.7	3.21	3.56
Fringe	63.8	83.4	103.0	3.26	3.27
Medium metro	69.8	86.4	102.9	2.77	2.75
Small metro	63.5	79.4	95.4	2.65	2.66
Nonmetro	25.8	33.4	39.9	1.26	1.07
Urbanized					
Adjacent to MSA	36.2	46.6	54.7	1.73	1.36
Remote	50.7	64.9	77.5	2.37	2.10
Less urbanized					
Adjacent to MSA	16.9	22.0	26.5	0.84	0.76
Remote	21.2	27.7	32.9	1.09	0.87
Rural					
Adjacent to MSA	5.8	7.6	10.1	0.30	0.41
Remote	6.3	9.3	11.0	0.49	0.29

*Excluding Alaska.
Note: Metro = metropolitan; MSA = Metropolitan Statistical Area.

The classification of internal medicine subspecialists as primary care physicians reduced urban–rural differences in the specialist ratio (see above). However, the inclusion of specialist residents increased the differences. During the late 1970s and 1980s about 17% of the specialists in metro areas were in residency training, in contrast to only 3% of those in nonmetro areas. The inclusion of residents is appropriate in view of their role in urban teaching hospitals.

Osteopaths

The number of osteopaths also grew rapidly between 1976 and 1987, increasing by 4.2% per year from 13 700 to 21 800. The osteopath/population ratio rose from 6.3 to 9.0 during the same period (Table 4). Urban–rural differences in the supply of osteopaths were small in comparison with the differences in the supply of allopaths. However, osteopaths were distributed across the urban–rural continuum in a distinct pattern, with the greatest supply concentrated in the most urban and most rural areas of the country (Figure 3).

The supply of osteopaths increased more rapidly after 1981 (Table 4). Some areas (nonmetro urbanized remote and less urbanized adjacent counties) actually lost osteopaths during the late 1970s. In contrast, all areas gained osteopaths after 1981, with the fastest growth occurring in the most urban and most rural areas.

Composition of the Physician Supply

The uneven distribution of primary care physicians, specialist physicians, and osteopaths across the urban–rural contin-

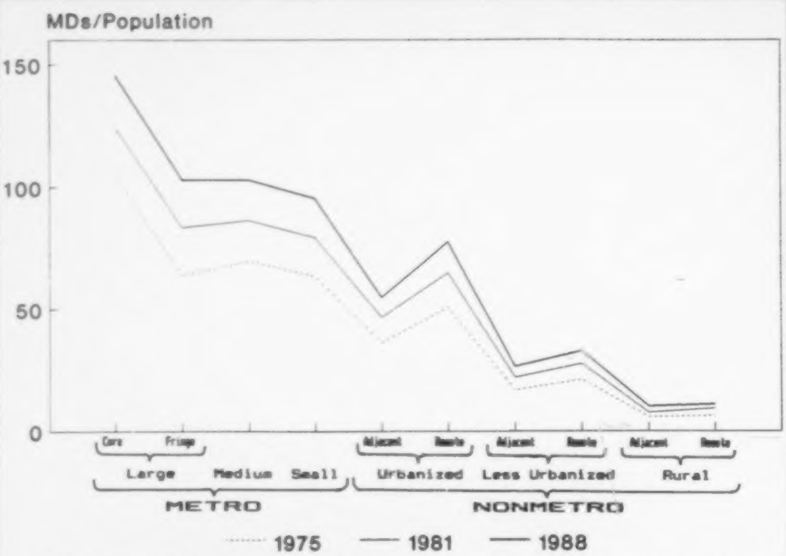

FIGURE 2—Nonfederal Specialist Physicians per 100 000 Persons by Urban–Rural Continuum Category, 1975–1988

uum produced major differences in the composition of the physician supply (Table 5). Primary care physicians represented a larger share of the physician supply in more rural areas, accounting for more than two thirds of the practitioners in nonmetro rural counties. In contrast, the proportion of specialist physicians was greater in more urban areas. The higher degree of physician specialization in more urban areas reflected the concentration of medical referral centers in cities and larger towns.

Osteopaths represented less than 5% of the physician supply during the late 1970s and 1980s (Table 5). However, they provided a disproportionate share of the physicians in more rural areas because they were relatively more willing to locate in nonmetro, less urbanized and rural counties than allopaths (Figures 1–3). By 1987 to 1988 more than 10% of the physicians in nonmetro rural counties were osteopaths. The role of osteopaths was even greater in eight states (Arizona, Florida, Michigan, Missouri, New Jersey, Ohio, Pennsylvania, and Texas) where licensing legislation was historically most favorable to the osteopathic profession.[17] In these states osteopaths accounted for 36% of the physicians in nonmetro rural counties.

The increasing specialization of the physician population had a direct impact on the composition of the physician supply. From 1975 to 1976 and 1987 to 1988 the proportion of primary care physicians declined, and the proportion of specialist physicians increased in every category of the urban–rural continuum (Table 5). The increase in the proportion of specialists was greatest in nonmetro counties.

Discussion

The evidence examined here indicates that the supply of nonfederal physicians increased in every part of the country. By the late 1980s all areas had more primary care and specialist physicians and osteopaths than in the mid-1970s. The rapid growth of the US physician supply was thus associated with the movement of more practitioners into predominantly rural areas, as well as urbanized rural and urban areas. However, the physician supply increased more rapidly in metro counties and nonmetro urbanized remote counties than in other nonmetro areas. As a result, urban–rural differences in the availability of physicians widened over time.

A major reason for the slow growth of the physician supply in predominantly rural areas may have been the limited number of places large enough to support physicians. Only 17% of the population of nonmetro rural counties lived in places with 1000 or more inhabitants in 1980.[18] The remainder of the population was dispersed in communities seemingly too small to support physicians, despite the spread of practitioners into more rural areas. However, this analysis was not designed to reveal the size of places within counties that actually gained physicians.

Osteopaths played a surprisingly important role in predominantly rural areas because they were relatively more likely to locate there than allopaths. As a result, osteopaths accounted for a disproportionate share of the physician supply in nonmetro rural counties, particularly in the eight states historically most hospitable to the profession. The factors that account for the greater willingness of osteopaths to locate in predominantly rural areas de-

TABLE 4—Nonfederal Active Osteopaths per 100 000 Resident Population by Urban–Rural Continuum Category, 1976–1987[a]

Urban–Rural Continuum Category	Active Osteopaths/Population Ratio			Mean Annual Change in Active Osteopath/Population Ratio	
	1976	1981	1987	1976–1981	1981–1987
US Total	6.3	7.0	9.0	0.13	0.33
Metro	6.9	7.7	9.8	0.17	0.35
Large metro					
Core	7.4	8.3	10.7	0.19	0.40
Fringe	7.7	8.6	10.5	0.17	0.32
Medium metro	6.5	7.5	9.6	0.19	0.35
Small metro	4.5	4.6	6.1	0.02	0.25
Nonmetro	4.6	4.7	6.2	0.03	0.24
Urbanized					
Adjacent to MSA	4.3	4.7	6.2	0.09	0.25
Remote	3.5	3.4	4.9	−0.01	0.25
Less urbanized					
Adjacent to MSA	4.7	4.7	5.7	0.00	0.16
Remote	5.2	5.4	7.0	0.04	0.27
Rural					
Adjacent to MSA	3.8	4.0	4.8	0.05	0.13
Remote	5.6	5.6	8.3	0.00	0.44

[a]Excluding Alaska.

Note: Metro = metropolitan; MSA = Metropolitan Statistical Area.

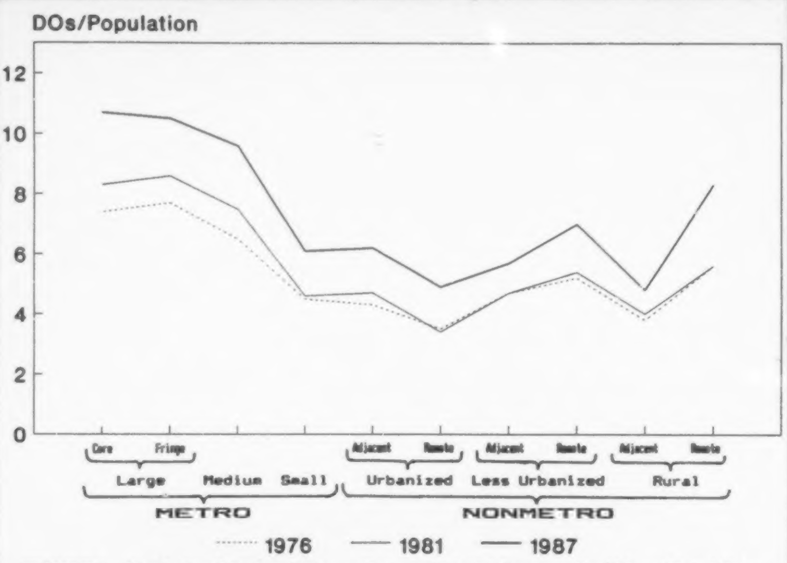

FIGURE 3—Nonfederal Active Osteopaths per 100 000 Persons by Urban–Rural Continuum Category, 1975–1988

serve further investigation in view of the reluctance of allopaths to practice in the same locations.

Despite the slow growth of the physician supply in predominantly rural areas, all nonmetro areas gained primary care physicians more rapidly during the 1980s than before. The faster growth may have been due to two different factors. First, increasing competition among physicians in metro areas may have encouraged more practitioners to select nonmetro locations. The slower growth of the primary care physician supply in most metro areas after 1981 supports this interpretation. Second, the number of general practice physicians decreased more slowly during the 1980s than the 1970s, elevating the net growth of the primary care physician supply.[19] The impact was greatest in nonmetro areas, where general practitioners represented a larger share of primary care physicians.

TABLE 5—Percent Composition of the Nonfederal Physician Population by Urban–Rural Continuum Category, 1975–1976 and 1987–1988[a]

	Percent of Nonfederal Physician Population[b]					
	1975–1976			1987–1988		
Urban–Rural Continuum Category	Primary Care Physicians	Specialist Physicians	Active Osteopaths	Primary Care Physicians	Specialist Physicians	Active Osteopaths
	(%)	(%)	(%)	(%)	(%)	(%)
US Total	45.1	50.2	4.7	44.0	51.4	4.6
Metro	43.6	52.0	4.4	42.8	52.8	4.4
Large metro						
Core	43.5	52.8	3.7	42.6	53.5	3.9
Fringe	43.8	50.0	6.1	43.0	51.7	5.3
Medium metro	43.7	51.4	4.9	43.1	52.1	4.9
Small metro	43.3	52.9	3.8	42.9	53.7	3.4
Nonmetro	55.9	37.3	6.8	52.7	41.0	6.3
Urbanized						
Adjacent to MSA	50.9	43.8	5.3	47.3	47.3	5.4
Remote	46.0	50.4	3.5	45.7	51.1	3.2
Less urbanized						
Adjacent to MSA	62.0	29.6	8.4	57.9	34.7	7.4
Remote	59.6	32.3	8.1	56.4	35.9	7.7
Rural						
Adjacent to MSA	74.5	15.4	10.2	68.2	21.5	10.3
Remote	70.7	15.4	13.9	66.4	19.2	14.4

[a] Excluding Alaska.
[b] Percents may not sum to 100.0% because of rounding.
Note: Metro = metropolitan; MSA = Metropolitan Statistical Area.

Specialist physicians in nonmetro areas were affected by different factors than primary care physicians because most nonmetro areas gained specialists more slowly after 1981. One possible factor may have been the limited number of places large enough to support specialists. By 1980 more than 36 million nonmetro residents lived outside places with 2500 or more persons in communities that the RAND studies showed rarely had specialists. However, more than 86% of this population lived in open country or places with fewer than 1000 persons that may never be able to support specialists.[18]

During the late 1970s and 1980s the physician supply in all areas also became more specialized. This trend was particularly dramatic in nonmetro rural counties, where the number of primary care physicians per specialist dropped from nearly five in 1975 to slightly more than three in 1988. The impact on the supply of medical services was unclear because specialists in rural areas may have spent much of their time providing primary care. The growth in the number of rural specialists also raised questions about the size of the case load needed to maintain physician expertise in specialized diagnostic and therapeutic procedures.[10]

Another important trend in nonmetro areas was the growing prominence of nonmetro urbanized remote counties as local centers of the physician supply. The rapid growth of the physician/population ratio and the higher proportion of specialists in these counties reflected the continuing development of secondary medical centers providing a variety of health services for rural areas some distance from major cities.[20] These centers were distinct from the tertiary medical centers in metro areas providing more specialized services for larger regions. The closing of hospitals in smaller rural communities and the greater competition from metro physicians in areas closer to cities may have contributed to the increasing concentration of physicians in rural secondary medical centers.

Nonmetro, less urbanized remote counties also became local centers of the primary care physician supply. These centers had fewer primary care physicians than nonmetro urbanized remote counties and did not have many specialist physicians. As a result, they appeared to be rural primary medical centers serving areas some distance from larger towns.

The most striking trend in metro areas was the continued rapid growth in the physician supply. It remains unclear whether this trend has produced a physician surplus because the demand for physician services depends on a complex variety of factors, including the costs of medical care, changes in health insurance coverage, advances in medical technology, the supply of midlevel health practitioners, and shifts in health care needs.

The even more rapid growth of the physician supply in large MSAs also deserved attention. By 1988 the suburbs of large MSAs had more primary care and specialist MDs than medium or small MSAs, reversing earlier patterns. The continued rapid growth was possible in part because physicians in large MSAs spent less time in patient care activities than those in other locations, restraining the actual growth in the supply of physician services.[22]

The variations in physician–population ratios described in this report do not necessarily reflect unequal access to physician services. Because many rural residents travel to urban areas to visit physicians, the average patient load per physician in rural areas is smaller than physician–population ratios suggest.[21] Rural physicians also work longer hours and see more patients than urban physicians, and this pattern reduces urban–rural differences in the supply of physician services.[22] Nevertheless, access to physician services outside densely populated urban areas still depends to a considerable extent on the location of physicians. In addition, rural communities have higher proportions of the poor and elderly than urban areas and may therefore require more physicians to meet local medical needs.[23]

Programs to provide more physician services in underserved areas need to take account of the fact that the most rural areas of the country remain unattractive to physicians. The present distribution of physicians in nonmetro areas suggests that it may be worth considering measures to encourage practitioners already located in rural primary and secondary medical centers to provide more care in surrounding areas. Some of the mechanisms that might be used to expand medical services into surrounding areas from these centers include mobile and satellite clinics, an increased role for midlevel health practitioners, and special transportation arrangements for nonemergency patients. ☐

Acknowledgments

The views expressed in this paper are solely those of the author. Laarni Dacquel provided expert research assistance. Valuable comments on an earlier version were received from Linda Swanson, Elaine Power, and Leah Wolfe. This research was supported by the Economic Research Service, US Department of Agriculture.

References

1. Ernst RL, Yett DE. *Physician Location and Specialty Choice.* Ann Arbor, Mich: Health Administration Press; 1985.
2. US Senate Special Committee on Aging. *The Rural Health Care Challenge.* Washington, DC: US Government Printing Office; 1988.
3. Rosenblatt RA, Moscovice IS. *Rural Health Care.* New York: John Wiley & Sons Inc; 1982.
4. US Congress, Office of Technology Assessment. *Health Care in Rural America.* Washington, DC: US Government Printing Office; 1990.
5. Graduate Medical Education National Advisory Committee. *Report to the Secretary, Department of Health and Human Services.* Hyattsville, Md: Health Resources Services Administration; 1980.
6. Schwartz WB, Newhouse JP, Bennett BW, Williams AP. The changing geographic distribution of board-certified physicians. *N Engl J Med.* 1980;303(18):1032–1037.
7. Newhouse, JP, Williams AP, Bennett BW, Schwartz WB. Where have all the doctors gone? *JAMA.* 1982;247(17):2392–2396.
8. Newhouse JP, Williams AP, Bennett BW, Schwartz WB. Does the geographical distribution of physicians reflect market failure? *Bell J Econ.* 1982;13:493–505.
9. Williams AP, Schwartz WB, Newhouse JP, Bennett BW. How many miles to the doctor? *N Engl J Med.* 1983;309(16):958–963.
10. Newhouse JP. Geographic access to physician services. *Annu Rev Public Health.* 1990;11:207–230.
11. Fruen MA, Cantwell JR. Geographic distribution of physicians: past trends and future influences. *Inquiry.* 1982;19:44–50.
12. Dennis T. Changes in the distribution of physicians in rural areas of Minnesota, 1965–85. *Am J Public Health.* 1988;78(12):1577–1579.
13. Kindig DA, Movassaghi H. The adequacy of physician supply in small rural communities. *Health Aff.* 1989;8(2):63–76.
14. US Department of Health and Human Services, Office of Data Analysis and Management. *Diffusion and the Changing Geographic Distribution of Primary Care Physicians.* Washington DC: US Government Printing Office; 1983.
15. Many Federal health programs get significant funding hikes. *The Nation's Health.* 1990;20(12):1,6–8.
16. Butler M: *Rural–Urban Continuum Codes for Metro and Nonmetro Counties.* Washington DC: Agriculture and Rural Economy Division, Economic Research Service, US Department of Agriculture; 1990.
17. American Osteopathic Association. *Yearbook and Directory of Osteopathic Physicians, 1990.* Chicago, Ill: American Osteopathic Association; 1990.
18. US Department of Commerce, Bureau of the Census. *1980 Census of Population. Volume 1; Characteristics of the Population.* Washington, DC: US Government Printing Office; 1983.
19. American Medical Association. *Physician Characteristics and Distribution in the U.S.* 1989 ed. Chicago, Ill: American Medical Association; 1989.
20. Brown DL. The redistribution of physicians and dentists in incorporated places of the upper Midwest, 1950–1970. *Rural Sociology.* 1974;39(2):205–223.
21. Kleinman JC, Makuc D. Travel for ambulatory medical care. *Med Care.* 1983;21(5):543–555.
22. Gonzalez ML, Emmons DW. *Socioeconomic Characteristics of Medical Practice, 1988.* Chicago, Ill: American Medical Association; 1989.
23. Rowland D, Lyons B. Triple jeopardy: rural, poor, and uninsured. *Health Serv Res.* 1989;23(6):975–1004.

Cognitive Impairment and Hospital Use

ABSTRACT

Background. An increasing number of older people are at higher risk of developing Alzheimer's Disease or another dementias. The resultant cognitive impairment has been well identified as one of the risk factors for nursing home placement but it has not been well studied as a risk for hospitalization.

Methods. To study the association between cognitive impairment and hospital use, this study examined data from a randomly selected cohort of community dwelling California elderly (California Senior Survey). The cohort was followed for one year (N = 940).

Results. Only about 17 percent of the study population was in the mildly or moderately/severe impaired category. Those participants with cognitive impairment were almost twice as likely as those without any impairment to be hospitalized and to be hospitalized for six days or more.

Conclusions. Although the cause of the cognitive impairment and hospitalization was not determined in this study, the data support the importance of cognitive impairment as a risk factor for hospital use even after controlling for age, ADL, IADL, and prior hospitalizations. The causes for this need to be further examined so the burden of such care can be reduced. (*Am J Public Health* 1991;81:1153–1157.)

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Introduction

Cognitive status has been identified as one of the risk factors for nursing home placement[1] but it has not been well studied as a cause of hospitalization. Although several studies have looked at health services utilization due to Alzheimer's Disease or other dementias,[2–4] these studies have been mainly interested in estimating costs of care, particularly cost of nursing home care.[4] Further, they have relied largely on cross-sectional data sources and the number of individuals with cognitive impairment in some of these data sets has been small.

Few studies have looked at the hospitalization admission rate and length of stay in a random sample of community dwelling elderly based on their cognitive status. Huang and Hu[2] found that the elderly demented utilized an average of 4.81 days of short-term acute hospital care per person. This represented an additional utilization of short stay hospitals due to Alzheimer's Disease of 0.81–1.02 days per year based on the national hospital discharge survey data. Coughlin and Liu,[5] using data from the 1981–82 National Long-Term Care Channeling Demonstration Project and the Short Portable Mental Status Questionnaire (SPMSQ) to rate a person's cognitive status, found that the severely cognitively impaired spent a higher proportion of survival days in hospitals (.10) then mild/no cognitive impaired (.08) after six months of follow-up. More recently, Binder and Robins[6] found that community dwelling elderly were more likely to be hospitalized if they scored poorly on the Folstein Mini-Mental State Exam.

In response to the limited research on this topic, the purpose of the present study was to examine the relation between cognitive status and hospitalization in a randomly selected cohort of community dwelling Medicaid recipients. Additionally, the study examined the relation of cognitive status and length of hospital stays. The cohort was followed for one year.

Methods

Design

The data were drawn from the California Senior Survey (CSS), a random sample of community dwelling elderly MediCal (Medicaid) recipients from eight representative communities within California. The CSS respondents were randomly selected from files which contained the names, addresses, and other basic demographic data with all MediCal recipients. Only MediCal recipients 65 years of age and older were sampled. CSS respondents were interviewed face to face in their homes in 1982. Comparisons between the survey data and statewide data indicated that respondents were typical of California's MediCal elderly population.[7] More complete descriptions of the sampling methodology and sample characteristics have already been published.[7,8]

Hospital utilization data were obtained for one year following the inter-

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TABLE 1—Selected Characteristics of Study Cohort by Level of Cognitive Impairment (percentage distribution) N = 940

	Not Impaired	Mildly Impaired	Mod/Sev Impaired
(Total %)	(8[illegible]%)	(9.3%)	(7.4%)
Sex			
Males	89.3	5.4	5.3
Females	81.0	10.8	8.3
Age (years)			
65–74	91.1	6.1	2.8
75–84	81.2	10.1	8.7
85+	67.2	15.7	17.2
Marital Status			
Married	82.9	8.3	8.8
Widowed	81.4	11.0	7.6
Separated/Divorced	88.7	6.4	5.0
Single	89.1	3.6	7.3
Living Arrangement			
Alone	87.6	8.5	4.0
With Others	78.0	10.4	11.6
Race/Ethnicity			
White	86.3	7.4	6.3
Hispanic	78.7	13.1	8.2
Black	79.1	10.8	10.1
Asian	79.4	11.2	9.4
Education			
None	51.7	20.0	28.3
Elementary	83.3	9.3	7.3
High School	88.6	5.9	5.6
Beyond High School	84.8	11.2	3.9

view. The hospital utilization data came from MediCal paid claims and computer tapes which were merged with data obtained from Medicare intermediaries.[9] Combining these two sources provided data on number of days hospitalized for 92 percent of the original CSS sample (N = 1,037). To assure accuracy, the computer tapes were examined for any bills submitted for hospitalization during the follow-up year to either MediCal or Medicare intermediaries on behalf of CSS respondents. To overcome any problems that may have been caused by late billing, bills submitted up to two years following the interview were reviewed.

Besides those without hospital data, 15 other respondents were also dropped from the present study. Six lacked cognitive status data and nine had died within a few months of the survey and so their hospital data were necessarily censored. Analyses were run with and without these six respondents and they did not significantly affect the outcome. However, dropping these six respondents from the analyses was considered the more appropriate method. Although 25 other respondents also had died within a year of the baseline survey, they all had extensive hospital stays before their death and so it was possible to properly classify their hospital use for the present analyses.

No significant differences in terms of major sociodemographic and health status characteristics were noted between survey respondents with and without complete data. All respondents with complete hospital and cognitive status data were used for the study analyses (N = 940).

A structured measure of mental status was administered, the Short Portable Mental Status Questionnaire (SPMSQ). This instrument has high validity and test-retest reliability.[10] The widely used test is a 10-item questionnaire that is designed to measure several areas of intellectual domain including orientation, short- and long-term memory, general information, and problem solving. Pfeiffer[10] originally recommended adjustments for race and education. However, more recent versions of the SPMSQ only adjust for education.[11,12] Thus the present analysis chose to only adjust SPMSQ scores for education, although both race and education were entered as covariates in the multiple regression analysis.

Using Pfeiffer's recommendations, the SPMSQ scores used to categorize participants were: not impaired (0–2 errors), mildly impaired (3–4 errors), and moderately to severely impaired (over 4 errors).[10] The SPMSQ was administered in the elderly person's home by trained interviewers who also administered a comprehensive questionnaire composed of several previously described instruments.[13] Included in this battery were two commonly used functional status scales: the Activity of Daily Living (ADL) and Instrumental Activity of Daily Living (IADL) scale.[14,15]

Two dichotomous variables measuring hospital utilization were used: whether a respondent had been hospitalized within the year following the interview, and whether a respondent experienced a hospital stay of six or more days in the year following the interview. The figure of six days was selected because it was slightly higher than the average length of stay for all respondents (mean length of stay = 5.2 days).

Analysis

The present analyses used bivariate analyses (Chi-square) and odds ratios. A series of logistic regression models were used to examine the relation of the levels of cognitive status with the two indices of subsequent hospital utilization. Age, education, race, and prior hospitalizations during the year prior to the survey were controlled. The statistical program used was SAS and analyses were conducted on an IBM 3090 mainframe computer.

Study Population

Reflecting the diversity of California's ethnic population, the sample included a sizable portion of Blacks (17 percent), Hispanic (13 percent), and Asians (12 percent), although a majority (58 percent) were White. Seventy-two percent of the sample were females and over 60 percent of the sample lived alone. The age distribution of the sample was 42 percent (65–74 years of age), 44 percent (75–84 years old), and 14 percent (85 years old and over). The majority of participants were widowed (56 percent) with 23 percent being married, 6 percent single, and 15 percent separated or divorced. The educational level obtained by the participants was 33 percent finished high school and 19 percent had an education beyond high school, 6 percent had no formal education, and about 42 percent finished elementary school.

Results

Table 1 gives selected characteristics of the participants by level of cognitive impairment. Overall about 17 percent of the study population was either mildly or moderately to severely impaired. There was a higher prevalence of cognitive impairment in females than males. The prevalence of cognitive impairment increased significantly with age. Marital status was not related to cognitive impairment but cognitive impairment was more prevalent in those living with others than in those living alone. There were no race or ethnicity differences in the prevalence of cognitive impairment, but lack of education was associated with cognitive impairment.

There was a significantly higher prevalence of cognitive impairment in those participants with two or more ADL dependencies. Similarly, an increased number of instrumental activities of daily living (IADL) dependencies was also significantly associated with cognitive impairment with money management having the strongest association (Table 2). The number of total chronic conditions was not significantly related to cognitive impairment (Table 3). Arthritis was negatively associated with cognitive impairment whereas stroke was positively related.

TABLE 2—Self-care Capacity of Study Cohort by Level of Cognitive Impairment (percentage distribution) N = 940

	Not Impaired	Mildly Impaired	Mod/Sev Impaired
(Total %)	(83.3%)	(9.3%)	(7.4%)
ADL Dependencies			
0	88.2	7.4	4.4
1	85.1	8.9	6.1
2+	61.1	16.8	22.1
IADL Dependencies			
0	92.5	6.5	0.9
1	95.2	3.7	1.1
2	88.3	7.8	3.9
3	80.3	12.2	7.5
4	82.4	9.8	7.8
5+	55.4	18.9	25.8

TABLE 3—Co-morbidity of Study Cohort by Level of Cognitive Impairment (percentage distribution) N = 940

	Not Impaired	Mildly Impaired	Mod/Sev Impaired
(Total %)	(83.3%)	(9.3%)	(7.4%)
Chronic Conditions			
Arthritis	86.3	7.4	6.3
Stroke	69.4	10.2	20.4
High Blood Pressure	84.9	8.4	6.7
Heart Condition	81.6	9.9	8.5
Respiratory	87.3	6.4	6.4
Gastrointestinal	85.6	7.1	7.4
Diabetes	82.9	8.6	8.6
Number of Conditions			
0	76.3	12.5	11.3
1	81.3	13.7	5.1
2	87.0	6.8	6.3
3	85.8	7.8	6.5
4+	81.8	8.5	9.7

Odds ratios adjusted for age, education, race, and any hospitalizations during year prior to survey are shown in Table 4. Those participants with cognitive impairment were more likely to be hospitalized and to have increasing length of hospitalizations. Similarly, greater impairments on the ADL and IADL scales were associated with increased risk of hospitalization.

The number of dependencies on the ADL and IADL was collapsed into categories to facilitate analyses. Very few respondents had more than two ADL dependencies and so a single dependency on the ADL was considered a mild ADL impairment whereas being unable to perform two or more ADL functions was considered to be a moderate to severe ADL impairment. Cut points for the IADL were selected to maximize the relationship between IADL and hospital use. One to two IADL dependencies were judged to be a mild IADL impairment, three to four a moderate impairment, and five or more IADL dependencies were classified as a severe dependency. Any level of impairment on the SPMSQ was predictive of hospital use. Increased impairment on any of the scales was associated with increased risk of extended hospital stays. Further, Table 4 illustrates that those elderly with severe IADL impairments are especially at increased risk of longer hospitalizations. Mild impairment on the ADL was not generally associated with increased risk of hospital use.

Among the covariates, having been hospitalized during the year prior to the survey and being White were the only covariates related to subsequent hospital use. Previous hospitalization was the only covariate predictor of subsequent stays of six or more days (OR 2.73; CI = 1.96, 3.81).

To directly compare the SPMSQ with the ADL and IADL as predictors of hospitalization, additional logistic regression models were constructed. One set of models included both the SPMSQ and the ADL. Another set included the SPMSQ and the IADL (the ADL and IADL were too highly correlated to have included both of these functions in the same regression models). In these direct comparisons, the SPMSQ proved to be more predictive of future hospital use than the ADL but not as good as the IADL. Five or more dependencies on the IADL was an especially strong predictor of future hospital use even when controlling for moderate to severe impairments on the SPMSQ.

Discussion

The data are consistent with previous findings that cognitive impairment in community dwelling elderly is more common among females, older age groups, and those living with others.[16,17] The prevalence rates of cognitive impairment in studies of community elderly vary from a low of 1.6 percent (severe impairment) to a high of over 15 percent for the mildly impaired. The present study found an overall rate of 16.7 percent. The prevalence of cognitive impairment among the old old (85 years of age and older) was found to be higher (32.8 percent) than that previously reported (25 percent).[16] Two recent studies have also found higher than expected prevalence rates in the old old. Forty seven percent of those 85 years old

TABLE 4—Odds Ratios for Hospital Use by Level of Impairment

		Any Hospital Use		Hospitalized 6 Days or More	
	Percent With Impairment	Odds Ratio	(95% CI)	Odds Ratio	(95% CI)
ADLs					
Mild	26.7	1.30	(.92, 1.82)	1.42	(.97, 2.07)
Moderate/Severe	14.1	1.52	(.99, 2.33)	1.87	(1.19, 2.93)
IADLs					
Mild	45.0	1.52	(.87, 2.68)	2.95	(1.31, 6.64)
Moderate	26.8	2.04	(1.13, 3.69)	3.45	(1.50, 7.96)
Severe	16.8	3.48	(1.84, 6.55)	7.36	(3.12, 17.38)
SPMSQ					
Mild	9.3	1.85	(1.14, 2.98)	1.89	(1.14, 3.16)
Moderate/Severe	7.4	1.76	(1.03, 3.01)	2.10	(1.20, 3.68)

NOTE: Odds Ratios adjusted for age, education, race (White/non-White), and any hospitalizations during year prior to baseline survey.

These data categorize IADL dependencies as follows: 1-2 = mild; 3-4 = moderate; 7.5 = severe.

or older were found to have probable Alzheimer's disease in one study conducted in a geographically defined community in which 80.8% of the age-eligible residents were evaluated.[18] In another study of dementia in a retirement community, Pfeffer[19] found the prevalence rate of dementias to be 56.7 percent in those 85 years old and older. Although comparisons among various studies are difficult due to differing methods of case ascertainment, sampling methods and study populations, these data suggest that the public health impact of cognitive impairment in the elderly may be greater than previously described.

Our data do not support the concept of a racial or ethnic difference in cognitive impairment as implied in earlier suggestions for adjusted scoring of the SPMSQ.[10] The data did show a significant association between educational level and performance on mental status examinations. This association has been documented in other studies as well.[20] For example, Katzman[21] studied a probability sample of 5,055 community dwelling elderly in Shanghai. The Chinese Mini-Mental State (CMMS) was used to evaluate mental status. In comparison to those with formal education, this study found significantly lower scores on the mental status test as well as a different error pattern among the uneducated even after making adjustments for education. The reason for the association between educational level and suggested cognitive impairment has not yet been fully explained.[22]

Although the cause of the cognitive impairment and hospitalization were not determined in this study, the data indicate that cognitive impairment is a risk factor for hospital admissions and longer than average hospital stays even after controlling for age, ADL, IADL, and prior hospitalizations. The longer length of stay may be due to lack of appropriate posthospital placement or to complications of hospitalization.[23,24] Whatever the reasons for increased hospital use, the increasing prevalence of cognitively impaired elderly will result in an enormous financial burden for the patient, family, and government and has significant public health consequences.[25,26] □

Acknowledgments

The data used in this analysis come from the California Senior Survey, which was the comparison group for the California Multipurpose Senior Services Project (MSSP), a US Health Care Finance Administration (HCFA 115) research and demonstration project (Grant #11-P-07553).

This project was funded in part by the California Academic Geriatric Resource Program.

References

1. Branch LG, Jette AL: A prospective study of long-term care institutionalization among the aged. Am J Public Health 1982; 72:1373–1379.
2. Huang L, Hu T: Economic costs of senile dementia in the United States. National Institute on Aging, Control No. N 01-AG-3-2123. Washington, DC: Applied Systems Institute, 1983.
3. Hay JW, Ernst RL: The economic costs of Alzheimer's disease. Am J Public Health 1987; 77:1169–1175.
4. Coughlin T, Liu K: Health care costs of dementia: A review. Washington, DC: Health Policy Center, Urban Institute, September 1987.
5. Coughlin T, Liu K: Health care costs of older persons with cognitive impairments. Washington, DC: Urban Institute, January 1988.
6. Binder EF, Robins LN: Cognitive impairment and length of hospital stay in older persons. J Am Geriatr Soc 1990; 38:759–766.
7. Miller LS, Clark ML, Clark WF: The comparative evaluation of California's Multipurpose Senior Services Project (MSSP). Home Health Care Serv Q 1985; 6:49–79.
8. Clark WF, Pelham AO, Clark ML: Old and poor. Lexington, MA: Lexington Books, 1988.
9. Clark ML, Walter L, Miller LS: Estimates of medical services cost: MediCal and Medicare paid claims. Berkeley, CA: Multipurpose Senior Services Project (MSSP) Evaluation Unit, 1983.
10. Pfeiffer E: A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. J Am Geriatr Soc 1975; 23:433–441.
11. Pfeiffer E: The psychosocial evaluation of the elderly patient. *In*: Busse EW, Blazer DE (eds): Handbook of Geriatric Psychiatry. New York: Van Nostrand Reinhold, 1980.
12. Gallo JJ, Reichel W, Anderson L: Handbook of Geriatric Assessment. Rockville, MD: Aspen Publications, 1988.
13. Lubben JE: Health and psychological assessment instruments for community based long-term care: The California Multipurpose Senior Services Project (MSSP) experience. Doctoral dissertation, University of California, Berkeley, 1984.
14. Katz S, Ford AB, Moskokwitz RW, Jackson B, Jaffe MW: Studies of illness in the aged: The index of ADL: A standardized measure of biological and psychological function. JAMA 1963; 185:914–919.
15. Lawton MP, Brody EM: Assessment of older people: Self-maintaining and instrumental activities of daily living. Gerontologist 1969; 9:179–186.
16. Cross PS, Gurland BJ: The epidemiology of dementing disorders. Washington, DC: US Congress, Office of Technology Assessment, 1985.
17. Mortimer JA, Schuman LM, French LR: Epidemiology: Overview and prospects. *In*: Mortimer JA, Schuman LM (eds): The Epidemiology of Dementia. New York: Oxford University Press, 1981.
18. Evans DA, Funkenstein HH, Albert MS, Scherr PA, Cook NR, Chown MJ, Herbert LE, Hennekens CH, Taylor JO: Prevalence of Alzheimer's disease in a community population of older persons. JAMA 1989; 262:2551–2556.
19. Pfeffer RI, Afifi AA, Chance JM: Prevalence of Alzheimer's disease in a retirement community. Am J Epidemiol 1987; 125:420–436.
20. King H, Locke F: Health risk and lifestyle. *In*: Rothschild H (ed): Risk Factors for Senility. New York: Oxford University Press, 1984.
21. Katzman R, Zhang MY, Quang Ya Qu, Wang ZY, Liu WT, Yu E, Wong SC, Salmon DP, Grant I: A Chinese version of the Mini-Mental State Examination; impact of illiteracy in a Shanghai dementia survey. J Clin Epidemiol 1988; 41:971–978.
22. Berkman LF: The association between educational attainment and mental status examinations: Of etiologic significance for senile dementias or not? J Chronic Dis 1986; 39:171–175.

23. Johnston M, Wakeling A, Graham N, Stokes F: Cognitive impairment, emotional disorder and length of stay of elderly patients in a district general hospital. Br J Med Psychol 1987; 60:133–139.
24. Erkinjuntti T, Autio L, Wikstrom J: Dementia in medical wards. J Clin Epidemiol 1988; 41:123–126.
25. Weiler PG: The public health impact of Alzheimer's disease. Am J Public Health 1987; 77:1157–1158.
26. US Congress, Office of Technology Assessment: Losing a Million Minds: Confronting the Tragedy of Alzheimer's Disease and Other Dementias. Pub. No. OTA-BA-323. Washington, DC: Govt Printing Office, 1987.

The German Health Care System: What Can We Learn from It?

As health costs continue to rise and many Americans find themselves without adequate health care coverage, attention has turned to other countries for answers. In recent years, Canada's government-run "single-payer" system has been the subject of intense debate. In the past year, though, interest has grown in the German "all-payer" health care system. Could it become a model for reforming health care in the United States? The May issue of *The Internist: Health Policy in Practice*, published by the American Society of Internal Medicine, examines what health care lessons can be learned from the German experience.

Uwe E. Reinhardt, PhD, the James Madison Professor of Political Economy at Princeton University, writes that if organized American medicine wishes to get ready for health care by the end of the century, it will need to study the "all-player" system of Western Europe in which "all players in a market area are bound by common fee schedules and all providers are subject to overall budget constraints." Dr. Reinhardt, who is a member of the Physician Payment Review Commission, adds that a move toward an all-payer system in the United States would require the development of a mechanism in which payers, including the federal and state government, could jointly negotiate binding fee schedules with the providers of health care.

German economist J. Matthias Graf von der Schulenburg observes that the annual increase of per capita health care spending in Germany was one of the lowest among industrialized nations between 1980 and 1987 (followed only by Japan, which more or less copied the German health care system). More than 1100 "statutory sickness funds"—self-sustaining, nonprofit institutions generally organized by geographic location, place of employment, or trade or profession—provide care for the German population. Although Germany saw a sudden increase in health care costs in the mid-1970s, Dr. Graf von der Schulenburg examines how costs came under control through negotiations at the different levels (federal, state, and local) of the health care system.

Although Germany's health care system has had a good record in restraining costs and prices, there have been trade-offs in terms of physician supply and demand, says Dale Rublee, PhD, a policy analyst with the American Medical Association. The growth in supply has become the medical profession's greatest long-range concern and Germany's most visible problem, he says. Germany has the most medical students per capita (next to Italy) and has seen rapid growth of medical specialists. The trade-off with an oversupply of physicians has been an overall decrease in physician earnings in the past few years in Germany.

Robert B. Doherty writes in *Capital Currents* that recent studies reporting that a quarter of all health care spending goes to "administrative waste" is overstated and that the data, especially concerning overhead, need to be closely examined. Doherty, ASIM's vice president of governmental affairs and public policy, adds that one of the reasons overhead costs may seem to be higher in the United States than in other countries is that more dollars are invested in quality assurance (such as peer review organizations or continuing medical education requirements).

For a copy of the May issue of *The Internist: Health Policy in Practice*, send $3 to ASIM, 1101 Vermont Avenue, NW, Suite 500, Washington, DC 20005-3457.

Cause-Specific Mortality in a Population-Based Study of Diabetes

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ABSTRACT

Background. Mortality from vascular diseases has been reported to be high in diabetic persons.

Methods. To evaluate mortality from these and other specific causes, we examined cause-specific age-sex standardized mortality ratios in a geographically defined population of younger onset (diagnosed before age 30 and taking insulin, n = 1200) and older onset (diagnosed after age 30, n = 1772) diabetic persons followed for 8.5 years. Cause of death was determined from death certificates.

Results. In younger onset persons, age-sex standardized mortality ratios were significantly high ($P < .05$) for all causes of death (7.5) as well as for diabetes (191), all heart disease (9.1), ischemic heart disease (10.1), other heart disease (6.3), nephritis and nephrosis (41.2), accidents (2.9), and all other causes (3.2). In older onset persons, age-sex standardized mortality ratios were significantly high for all causes of death (2.0) as well as for diabetes (16.8), all heart disease (2.3), ischemic heart disease (2.3), other heart disease (2.1), stroke (2.0), and pneumonia and influenza (1.7).

Conclusions. Diabetic persons experience very high mortality, especially from vascular diseases, compared to the general population. (*Am J Public Health.* 1991;81:1158–1162)

Introduction

Diabetic persons have been shown to be at greater risk of mortality from cardiovascular disease,[1–18] stroke,[1,2,5,9,11] and renal disease[14,19] compared to nondiabetic persons. Risk estimates for cancer have been variable.[1,3,11,14,15,20] Many of these earlier results are based on clinic populations, include only people with type I or type II diabetes, include males only, or are based on small numbers of cases. Thus, the results of many of these studies may not be applicable to a wider diabetic population.

In the current investigation we examine data from a population-based study, the Wisconsin Epidemiologic Study of Diabetic Retinopathy. Cause-specific mortality from this sample of diabetic persons is compared to the general population.

Methods

The study population has been described in detail in previous reports.[21–23] The study area is composed of 11 counties in southern Wisconsin (1980 population of 839 324). Diabetic persons were identified by a review of the records of 452 of the 457 physicians providing primary care to diabetic persons in the year beginning July 1, 1979. Chart reviews were done on 9841 patients. Of these, 558 were excluded because they were confined to nursing homes, had died prior to July 1, 1979, had incorrect diagnostic codes, had moved, or had gestational diabetes.

A two-part sample of 2990 of the remaining 9283 patients was selected on July 1, 1980, for examination. The first part consisted of all persons diagnosed before age 30 and taking insulin, referred to as younger onset (n = 1210). The second part consisted of a probability sample of persons diagnosed as having diabetes after age 30 confirmed by a random or postprandial serum glucose of at least 11.1 mmol/L or a fasting serum glucose of at least 7.8 mmol/L on at least two occasions, referred to as older onset (n = 1780). The specifics of the sampling strategy and exact proportions appear in an earlier publication, but approximate proportions were 25% for eligible persons with 0 to 4 or 5 to 14 years duration and 100% for persons with 15 or more years of diabetes.[23] Of the older onset group, 824 were taking insulin and 956 were not. Because it was not possible to examine the entire population, these sample sizes were chosen to ensure that an adequate number of cases of diabetic retinopathy would be found to evaluate risk factors for this complication.

Of the original sample, 18 (10 younger onset and 8 older onset) had died or were lost prior to July 1, 1980. These persons were excluded from further analysis. The remaining persons were contacted annually by telephone to determine vital status. In addition, relatives, designated contact persons, and physicians were contacted, and newspaper obituaries were reviewed daily. An annual request was made to the Wisconsin Center for Health Statistics, Section of Vital Statistics, for death certificate information. Wisconsin death rec-

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This paper was submitted to the journal November 21, 1990, and accepted with revisions February 19, 1991.

ords through 1988 have been searched. Finally, information on persons who moved out of Wisconsin and were suspected of being deceased and persons lost to follow-up was submitted to the National Death Index at the National Center for Health Statistics for matching against national death data through 1987.

The study period was from July 1, 1980, through December 31, 1988. Only deaths that had been confirmed by a death certificate were included in the analysis. Persons who were thought to be deceased but for whom a death certificate could not be located were considered to be alive as of the last contact date they were known to be alive. An analysis was undertaken at this time because nearly half of the older onset group had died, affording adequate deaths to examine a number of underlying causes. While the number of deaths in the younger onset group is considerably smaller and should probably be regarded as preliminary, it is still large enough to investigate the major causes of death.

All medical conditions on the Wisconsin death certificate were coded by trained nosologists in the Wisconsin Division of Health using the International Classification of Diseases, Ninth Revision (ICD-9). The underlying cause of death was selected by the Automated Classification of Medical Entities computer program.[24,25] Out-of-state certificates were coded and processed in the same manner. The cause-specific mortality analysis of the present investigation was based on the underlying cause of death.

Standardized mortality ratios (SMR) were used to compare the mortality experience of the two diabetes groups with that of the general Wisconsin population.[26] The Wisconsin rather than the US population was used as the standard because it is closer in racial and ethnic composition to the diabetic population, which is predominately non-Hispanic White. Age-, sex-, and cause-specific deaths and age- and sex-specific population estimates for the standard population were obtained from published tables.[27,28]

Results

Males comprised 52.2% of the younger onset group and 45% of the older onset group. Mean age at diagnosis and standard deviation (SD) in the younger onset group was 14.6 ± 7.6 years. In the older onset group, mean age ± SD at diagnosis was 55.5 ± 12.6 years. Mean age ± SD at the start of the study was 28.4 ± 13.3 and 66.7 ± 11.5 years in the younger and older onset groups, respectively.

TABLE 1—Vital Status of Younger and Older Onset Diabetic Persons, Wisconsin Epidemiologic Study of Diabetic Retinopathy, 1980–1988

	Younger Onset		Older Onset	
Status	n	%	n	%
Confirmed dead	145	12.1	859	48.5
Suspected dead[a]	5	0.4	17	1.0
Alive at last contact	1039	86.6	883	49.8
Lost to follow-up[b]	11	0.9	13	0.7
Total	1200	100.0	1772	100.0

[a] Treated as alive as of last contact at which known to be living.
[b] No contact for more than 2 years.

The vital status of the younger and older onset groups is presented in Table 1. There have been 145 (12.1%) and 859 (48.5%) confirmed deaths in the younger and older onset groups, respectively. Only 5 (0.4%) younger onset and 17 (1%) older onset deaths have not been confirmed. In addition, only 11 (0.9%) younger onset and 13 (0.7%) older onset persons have been lost to follow-up.

Table 2 presents the distribution of underlying cause of death among diabetic persons for the 12 most common causes of death in Wisconsin. In the younger onset group, diabetes was the leading underlying cause of death, accounting for 44.1% of all deaths. This was followed by heart disease (30.3%) and accidents (6.2%). In the older onset group, heart disease (48.8%) was the leading underlying cause of death, followed by diabetes (15.3%), malignant neoplasms (9.9%), and cerebrovascular disease (9.5%).

Standardized mortality ratios (SMR) by cause of death in the younger onset group are shown in Table 3. Mortality from all causes was 7.5 times more than expected from the general Wisconsin population and was more in excess in females (8.9) than in males (6.8). Among specific underlying causes of death, mortality from diabetes was 191 times greater than that expected. The SMR for heart disease was 9.1, with the excess being greater in females (10.3) than in males (8.7). The difference was more pronounced for ischemic heart disease (SMR of 13.5 in females, 9.1 in males). However, excess mortality for other heart disease was greater in males (7.3) than in females (4.5). There was also a sex difference with respect to mortality from accidents. The SMR for females (5.9) was significantly different from unity while in males it was not (2.0). The only other statistically significant excess mortality was due to nephritis and nephrosis (41.2). There were too few deaths from this cause to separate the sexes. Other SMRs, while substantially greater than unity, were not statistically significant, possibly due to a lack of power because of the small numbers of observed and expected deaths (Table 3).

Table 4 displays SMRs by cause of death in the older onset group. Mortality from all causes was twice the expected, and the excess was slightly greater in females (2.2) than males (1.9). As in the younger onset group, the greatest excess was due to diabetes (16.8). Mortality from heart disease was 2.3 times the expected and affected the sexes equally. Excess mortality due to ischemic heart disease was slightly higher in males while that due to other heart disease was higher in females. Cerebrovascular disease mortality was twice the expected. The only other statistically significant excess mortality was due to pneumonia and influenza (1.7).

Discussion

Many of the studies of cause-specific mortality among diabetic persons compared to nondiabetic persons are of clinic populations,[2,5,14,20] industrial or worker groups,[1,4,9,10,17] or other groups that are not population-based.[11] Clinic populations of diabetic persons may be biased in that they may be more likely to be suffering from complications of diabetes and have a poorer survival experience. Indeed, we have shown that in terms of all-cause mortality, diabetic persons with ocular diseases are at a disadvantage compared to those without.[29] On the other hand, studies based on worker groups may experience bias due to the "healthy worker" effect. Thus, population-based studies or studies based on a representative sample of a population offer the best opportunities for assessing the effect of diabetes on mor-

TABLE 2—Distribution of Cause of Death in Younger and Older Onset Diabetic Persons, Wisconsin Epidemiologic Study of Diabetic Retinopathy, 1980–1988

Cause of death	ICD-9 Codes	Younger Onset n	Younger Onset Cause of Death (%)	Older Onset n	Older Onset Cause of Death (%)
Malignant neoplasms	140–208	4	2.8	85	9.9
Diabetes mellitus	250	64	44.1	131	15.3
Heart disease	391–398,402,404–429	44	30.3	419	48.8
Ischemic heart disease	410–414	36	24.8	326	38.0
Other heart disease	391–398,402,404,415–429	8	5.5	93	10.8
Cerebrovascular disease	430–438	3	2.1	82	9.5
Atherosclerosis	440	0	0	10	1.2
Other arterial disease	441–448	0	0	6	0.7
Pneumonia and influenza	480–487	2	1.4	27	3.1
Chronic obstructive pulmonary disease	490–496	1	0.7	12	1.4
Chronic liver disease and cirrhosis	571	2	1.4	7	0.8
Nephritis and nephrosis	580–589	4	2.8	8	0.9
Accidents	800–949	9	6.2	12	1.4
Suicides	950–959	4	2.8	1	0.1
All other causes	—	8	5.5	59	6.9
Total	—	145	100.0	859	100.0

TABLE 3—Observed (O) and Expected (E) Deaths, Age- and Sex-standardized Mortality Ratios (SMR), and 95% Confidence Intervals (CI) by Cause of Death and Sex in Younger Onset Diabetic Persons, Wisconsin Epidemiologic Study of Diabetic Retinopathy, 1980–1988

Cause of Death (ICD-9 Code)	Male O/E	Male SMR	Male 95% CI	Female O/E	Female SMR	Female 95% CI	Total O/E	Total SMR	Total 95% CI
Malignant neoplasms (140–208)	—	—	—	—	—	—	4/5.1	0.8	0.2–2.0
Diabetes mellitus (250)	37/0.19	199.6	140.5–275.1	27/0.15	180.3	118.8–262.3	64/0.34	190.9	147.0–243.8
Heart disease (391–398, 402, 404–429)	31/3.6	8.7	5.9–12.3	13/1.3	10.3	5.5–17.7	44/4.8	9.1	6.6–12.2
Ischemic heart disease (410–414)	25/2.8	9.1	5.9–13.4	11/0.81	13.5	6.7–24.2	36/3.6	10.1	7.1–13.9
Other heart disease (391–398, 402, 404, 415–429)	6/0.82	7.3	2.7–15.9	2/0.44	4.5	0.5–16.3	8/1.3	6.3	2.7–12.5
Cerebrovascular disease (430–438)	—	—	—	—	—	—	3/0.74	4.1	0.8–11.8
Atherosclerosis (440)	—	—	—	—	—	—	0/0.05	0	—
Other arterial disease (441–448)	—	—	—	—	—	—	0/0.16	0	—
Pneumonia and influenza (480–487)	—	—	—	—	—	—	2/0.26	7.6	0.9–27.4
Chronic obstructive pulmonary disease (490–496)	—	—	—	—	—	—	1/0.40	2.5	0–13.9
Chronic liver disease and cirrhosis (571)	—	—	—	—	—	—	2/0.42	4.8	0.5–17.2
Nephritis and nephrosis (580–589)	—	—	—	—	—	—	4/0.10	41.2	11.1–105.5
Accidents (800–949)	5/2.4	2.0	0.7–4.8	4/0.68	5.9	1.6–15.2	9/3.1	2.9	1.3–5.5
Suicides (950–959)	—	—	—	—	—	—	4/1.4	2.8	0.8–7.3
All other causes	5/1.6	3.2	1.0–7.4	3/0.91	3.3	0.7–9.6	8/2.5	3.2	1.4–6.4
Total	89/13.1	6.8	5.5–8.4	56/6.3	8.9	6.7–11.5	145/19.4	7.5	6.3–8.8

Note. Sex-specific SMRs are not given where there are fewer than five total deaths.

tality. The present population-based study is notable for its substantial population of both younger and older onset diabetic persons of both sexes.

The population-based studies carried out in the United States have all found an excess mortality from cardiovascular disease.[3,6,7,12,16,18] These studies found SMR and relative risks (RR) in the range of 1.7 to 4.9. Although SMR and RR are not strictly comparable, they may give similar results in practice. The studies cited include both insulin- and noninsulin-dependent diabetic persons and thus are probably most comparable with our older onset group. In our older onset group we found an SMR of 2.3 for cardiovascular disease. This agrees remarkably well with the other population-based studies.[3,6,7,12,16,18] In addition, several of these studies note a differential effect of sex on cardiovascular disease

TABLE 4—Observed (O) and Expected (E) Deaths, Age- and Sex-standardized Mortality Ratios (SMR), and 95% Confidence Intervals (CI) by Cause of Death and Sex in Older Onset Diabetic Persons, Wisconsin Epidemiologic Study of Diabetic Retinopathy, 1980–1988

	Male			Female			Total		
Cause of Death (ICD-9 Code)	O/E	SMR	95% CI	O/E	SMR	95% CI	O/E	SMR	95% CI
Malignant neoplasms (140–208)	38/49.0	0.8	0.5–1.1	47/41.1	1.1	0.8–1.5	85/90.2	0.9	0.8–1.2
Diabetes mellitus (250)	49/3.2	15.4	11.4–20.4	82/4.6	17.6	14.0–21.9	131/7.8	16.8	14.0–19.9
Heart disease (391–398, 402, 404–429)	216/94.9	2.3	2.0–2.6	203/89.4	2.3	2.0/2.6	419/184.3	2.3	2.1–2.5
Ischemic heart disease (410–414)	179/73.2	2.4	2.1–2.8	147/65.8	2.2	1.9–2.6	326/139.0	2.3	2.1–2.6
Other heart disease (391–398, 402, 404, 415–429)	37/21.7	1.7	1.2–2.3	56/23.6	2.4	1.8–3.1	93/45.3	2.1	1.7–2.5
Cerebrovascular disease (430–438)	30/17.1	1.8	1.2–2.5	52/23.4	2.2	1.7–2.9	82/40.6	2.0	1.6–2.5
Atherosclerosis (440)	6/3.3	1.8	0.7–4.0	4/4.3	0.9	0.3–2.4	10/7.6	1.3	0.6–2.4
Other arterial disease (441–448)	3/3.6	0.8	0.2–2.5	3/2.2	1.4	0.3–4.0	6/5.8	1.0	0.4–2.3
Pneumonia and influenza (480–487)	14/8.7	1.6	0.9–2.7	13/7.6	1.7	0.9–2.9	27/16.3	1.7	1.1–2.4
Chronic obstructive pulmonary disease (490–496)	10/9.9	1.0	0.5–1.9	2/4.5	0.4	0–1.6	12/14.4	0.8	0.4–1.5
Chronic liver disease and cirrhosis (571)	5/1.9	2.6	0.8–6.2	2/1.2	1.7	0.2–6.2	7/3.1	2.3	0.9–4.7
Nephritis and nephrosis (580–589)	3/2.3	1.3	0.3–3.9	5/1.9	2.6	0.8–6.1	8/4.2	1.9	0.8–3.8
Accidents (800–949)	3/4.0	0.7	0.2–2.2	9/2.9	3.1	1.4–5.9	12/6.9	1.7	0.9–3.0
Suicides (950–959)	—	—	—	—	—	—	1/2.0	0.5	0–2.8
All other causes	31/21.6	1.4	1.0–2.0	28/24.4	1.1	0.8–1.7	59/46.0	1.3	1.0–1.7
Total	409/221.1	1.9	1.7–2.0	450/206.0	2.2	2.0–2.4	859/429.1	2.0	1.9–2.1

Note: Sex-specific SMRs are not given where there are fewer than five total deaths.

mortality in diabetic persons wherein females are at greater risk relative to nondiabetic persons than males.[6,7,12] However, this finding is not universal.[16,18] We also find no sex differential for either all heart disease or ischemic heart disease in the older onset group, but we do find one in the younger onset group, especially with respect to ischemic heart disease. However, in the Framingham study a trend is noted where the sex differential in cardiovascular disease incidence diminishes with advancing age.[6] If this effect carried over to mortality, it could explain our result.

Several studies have also investigated cerebrovascular disease mortality among diabetic persons.[1,2,5,9,11] Although none of these studies are population based, most of them found varying degrees of excess cerebrovascular disease mortality among diabetic persons. We found a significant twofold excess mortality from cerebrovascular disease in our older onset group. In the younger onset group there is a fourfold excess cerebrovascular disease mortality, but it does not reach statistical significance. However, the SMR is based on only three deaths.

Cancer was investigated in several studies with varying results.[1,3,11,14,15,20] In some studies cancer mortality did not differ from expected,[1,3,11,14] in one it was higher than expected,[15] and in one it was lower than expected in males only.[20] In two of these the results are based on small numbers of deaths,[1,14] and only two are population based.[3,15] We did not find that cancer mortality differed from the expected in either the younger or older onset groups.

Renal disease mortality has been found to be high in younger diabetic persons,[14] but the effect decreases sharply with age.[19] We also found excess mortality due to renal disease in the younger onset group. Mortality due to renal disease in the older onset group, while nearly double that expected, was not statistically significant. However, the real impact of renal disease on mortality in our population may be underestimated because some cases may also be included under diabetes as ICD-9 code 250.4, Diabetes with renal manifestations. If we add these to the other cases of renal disease, then the SMR for nephritis and nephrosis in the younger onset group is 72.1 (95% CI = 28.9–148.5), and in the older onset group it is 2.6 (95% CI = 1.3–4.7).

Two studies reporting on mortality from accidents and suicides combined, showed lower than expected mortality in males and higher than expected mortality in females.[2,14] In the younger onset group, we found higher than expected mortality from accidents in both males and females. In the older onset group, males had lower than expected and females had higher than expected mortality from accidents. In both groups only the result for females was statistically significant. Also, we found higher than expected suicide mortality in the younger onset group and lower than expected in the older onset group, but neither is statistically significant.

The only other significant result is higher than expected mortality due to pneumonia and influenza in the older onset group. We have not found this specific cause of death reported elsewhere. However, because we have a large number of deaths, especially in the older onset group, we are able to examine more specific causes of death than other population-based studies with smaller samples of diabetic persons. ☐

Acknowledgments

This research was supported by grant EY-03083 from the National Eye Institute (R. Klein). We are grateful to Fred Krantz, Wisconsin Center for Health Statistics, for providing death certificate information on Wisconsin residents, to Betty Sullivan and Peggy Peterson, Wisconsin Center for Health Statistics, for coding death certificates of out-of-state residents, to Stacy Meuer and Ane Weber for data processing, to Kathryn Linton and David DeMets for their helpful comments, and to Colleen Comeau and Luann Dohm for assistance with the manuscript.

References

1. Pell S, D'Alonzo CA. Factors associated with long-term survival of diabetics. *JAMA.* 1970;214:1833–1840.
2. Kessler II. Mortality experience of diabetic patients: a twenty-six year follow-up study. *Am J Med.* 1971;51:715–724.
3. Palumbo PJ, Elveback LR, Chu C-P, Connolly DC, Kurland LT. Diabetes mellitus: incidence, prevalence, survivorship, and causes of death in Rochester, Minnesota, 1945–1970. *Diabetes.* 1976;25:566–573.
4. Herman JB, Medalie JH, Goldbourt U. Differences in cardiovascular morbidity and mortality between previously known and newly diagnosed adult diabetics. *Diabetologia.* 1977;13:229–234.
5. Krolewski AS, Czyzyk A, Janeczko D, Kopczynski J. Mortality from cardiovascular diseases among diabetics. *Diabetologia.* 1977;13:345–350.
6. Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham study. *Diabetes Care.* 1979;2:120–126.
7. Heyden S, Heiss G, Bartel AG, Hames CG. Sex differences in coronary mortality among diabetics in Evans County, Georgia. *J Chron Dis.* 1980;33:265–273.
8. Jarrett RJ, McCartney P, Keen H. The Bedford Survey: ten year mortality rates in newly diagnosed diabetics, borderline diabetics, and normoglycaemic controls and risk indices for coronary heart disease in borderline diabetics. *Diabetologia.* 1982; 22:79–84.
9. Fuller JH, Shipley MJ, Rose G, Jarrett RJ, Keen H. Mortality from coronary heart disease and stroke in relation to degree of glycaemia: the Whitehall study. *Br Med J.* 1983;287:867–870.
10. Eschwege E, Richard JL, Thibult N, et al. Coronary heart disease mortality in relation with diabetes, blood glucose, and plasma insulin levels: the Paris Prospective Study, ten years later. *Horm Metabol Res.* 1983;15(suppl):41–46.
11. Fuller JH, Elford J, Goldblatt P, Adelstein AM. Diabetes mortality: new light on an underestimated public health problem. *Diabetologia.* 1983;24:336-341.
12. Barrett-Conner E, Wingard DL. Sex differential in ischemic heart disease mortality in diabetes: a prospective population-based study. *Am J Epidemiol.* 1983;118:489–496.
13. Reunanen A. Mortality in type 2 diabetes. *Ann Clin Res.* 1983;15(suppl 37):26–28.
14. Dorman JS, LaPorte RE, Kuller LH, et al. The Pittsburgh Insulin-dependent Diabetes Mellitus (IDDM) Morbidity and Mortality Study: mortality results. *Diabetes.* 1984; 33:271–276.
15. Green A, Hougaard P. Epidemiological studies of diabetes mellitus in Denmark: 5. mortality and causes of death among insulin-treated diabetic patients. *Diabetologia.* 1984;26:190–194.
16. Butler WJ, Ostrander LD Jr, Carman WJ, Lamphiear DE. Mortality from coronary heart disease in the Tecumseh study: long-term effect of diabetes mellitus, glucose tolerance, and other risk factors. *Am J Epidemiol.* 1985;121:541–547.
17. Pan W-H, Cedres LB, Liu K, et al. Relationship of clinical diabetes and asymptomatic hyperglycemia to risk of coronary heart disease mortality in men and women. *Am J Epidemiol.* 1986;123:504–516.
18. Kleinman JC, Donahue RP, Harris MI, Finucane FF, Madans JH, Brock DB. Mortality among diabetics in a national sample. *Am J Epidemiol.* 1988;128:389–401.
19. Geiss LS, Herman WH, Teutsch SM. Diabetes and renal mortality in the United States. *AJPH.* 1985;75:1325–1326.
20. Kessler II. Cancer mortality among diabetics. *J Natl Cancer Inst.* 1970;44:673–686.
21. Klein R, Klein BEK, Moss SE, DeMets DL, Kaufman I, Voss PS: Prevalence of diabetes mellitus in southern Wisconsin. *Am J Epidemiol.* 1984;119:54–61.
22. Klein R, Klein BEK, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy, II: prevalence and risk of diabetic retinopathy when age at diagnosis is less than 30 years. *Arch Ophthalmol.* 1984;102:520–526.
23. Klein R, Klein BEK, Moss SE, Davis MD, DeMets DL. The Wisconsin Epidemiologic Study of Diabetic Retinopathy, III: prevalence and risk of diabetic retinopathy when age at diagnosis is 30 or more years. *Arch Ophthalmol.* 1984;102:527–532.
24. National Center for Health Statistics. *Instruction Manual Part 2a. Instructions for Classifying the Underlying Cause of Death, 1989.* Hyattsville, Md: 1989. US Dept of Health and Human Services publication 240-95200045.
25. National Center for Health Statistics. *Instruction Manual Part 2c. ICD-9 ACME Decision Tables for Classifying Underlying Causes of Death, 1989.* Hyattsville, Md: 1989. US Dept of Health and Human Services publication 240-95200048.
26. Breslow NE, Day NE. *Statistical Methods in Cancer Research, Volume II. The Design and Analysis of Cohort Studies.* Lyon, France: International Agency for Research on Cancer, 1987.
27. Wisconsin Department of Health and Social Services, Division of Health. *Public Health Statistics.* Wisconsin Department of Health and Social Services. Madison, Wisc: 1980–1983.
28. Wisconsin Department of Health and Social Services, Division of Health. *Vital Statistics.* Wisconsin Department of Health and Social Services. Madison, Wisc: 1984–1988.
29. Klein R, Moss SE, Klein BEK, DeMets DL. Relation of ocular and systemic factors to survival in diabetes. *Arch Intern Med.* 1989;149:266–272.

Hypertension Management in Health Care for the Homeless Clinics: Results from a Survey

ABSTRACT

Background. With the exception of alcohol abuse, hypertension is the most common chronic physical health problem encountered among homeless persons. The material conditions of homelessness greatly complicate the management of this disorder. Some of the complications and their solutions are discussed here, based on the experiences of health clinics for the homeless in large US cities.

Methods. In 1988, the Stewart B. McKinney Homeless Assistance Act established health care clinics for homeless persons in 108 cities. We surveyed medical directors in these clinics, asking about the management of hypertension in this difficult-to-treat population; 65 responded.

Results. Comparisons between our survey data and those obtained in two recent surveys of clinicians in "normal" clinical practice provide interesting lessons in how medical practice is adapted to respond to the unique needs and problems of the urban homeless. Although therapeutic goals are similar, the means chosen to achieve them often are not.

Conclusions. The treatment of homeless hypertensives illustrates the problems inherent in strict biomedical models of disease and its alleviation. Preferred treatments, course of disease, and success of intervention are powerfully affected by social factors. (*Am J Public Health.* 1991;81:1163–1165)

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Introduction

With the exception of alcohol abuse, hypertension is the most common chronic physical health problem encountered among homeless persons, afflicting between 14% and 25% of this population.[1–4] The management of hypertension in the homeless is complicated by material and existential conditions. Proper dietary practice is exceedingly difficult to maintain,[5] rates of alcohol abuse exceed 50%,[4] storage and administration of medicines are difficult, and the cost of medication can be prohibitive. Compliance and maintenance of contact with homeless hypertensives are also difficult, and the asymptomatic stages of the disease produce little or no motivation to seek or maintain treatment.

In 1984, the Robert Wood Johnson Foundation, sensing the widespread unmet medical needs of the nation's homeless, funded health care for the homeless (HCH) clinics in 19 cities. In 1988, the McKinney Homeless Assistance Act extended the system to an additional 89 cities. We surveyed these 108 HCH clinics to learn how medical practice is adapted to the unique needs of homeless hypertensives, using a questionnaire loosely modeled on two recent surveys of hypertension management in "normal" clinical practice.[6,7] One of the clinics served only children and was dropped from the sampling frame; responses were obtained from 65 (60.7%) of the remaining 107 clinics.

Results

Initiation of Therapy

The Joint National Committee for the Detection, Education, and Treatment of High Blood Pressure (JNC)[8] recommends initiation of drug therapy when diastolic blood pressure (DBP) exceeds 94 mm Hg or at somewhat lower pressures if other risk factors are present; nondrug therapies are recommended for DBP between 90 and 94 mm Hg. HCH physicians largely adhere to the JNC recommendations. For an otherwise asymptomatic 45-year-old homeless white male, nondrug therapy would be initiated at an average DBP of 90.5 mm HG or at an average systolic blood pressure (SBP) of 151 mm Hg. Likewise, drug therapy would usually be initiated at a DBP of 97 mm Hg and at an SBP of 161 mm Hg. In both respects, HCH physicians differ little from clinicians in Maryland and New York City. Therapeutic goals are also very similar in both settings, with DBP <90 mm Hg as the target for the overwhelming majority.

The importance of factors other than blood pressure in deciding to initiate therapy was rated on a 1 (not important at all) to 4 (very important) scale. The most important other factor is target organ damage (mean rating [MR] = 3.88). (In a sample of homeless hypertensives being treated in the New Orleans HCH clinic, 20% had concurrent heart problems, compared to 2% of the normotensives.[2]) The co-occurrence of diabetes mellitus (MR = 3.79), elevated blood cholesterol (MR = 3.39), and being a smoker (MR = 3.32) were also rated as important factors in the decision to initiate therapy.

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This paper was submitted to the journal September 20, 1990, and accepted with revisions March 6, 1991.

TABLE 1—Percentage of Physicians Performing Various Laboratory Tests in the Initial Diagnostic Evaluation of a Recently Diagnosed Hypertensive Man

Laboratory Test	New York City	Maryland	HCH
Aldosterone	5	NR	0
Blood Urea Nitro	95	NR	85
Chest radiograph	100	70	34
CBC	100	67	66
Electrocardiogram	100	89	63
Intravenous pyel	19	11	5
Plasma renin	0	7	2
Serum cholesterol	95	NR	89
Serum creatinine	90	84	92
Serum potassium	100	82	86
Triglycerides	67	NR	55
Uric acid	90	NR	60
Urine culture	24	NR	0
Urinalysis	100	NR	94

Note. HCH = Health Care for the Homeless; NR = not reported.
Sources: New York City[7]; Maryland[6]; HCH, authors' survey.

Laboratory Protocols

Initiation of therapy usually implies a detailed laboratory workup to assess other risk factors and to establish baseline values.[8] Table 1 compares laboratory protocols of HCH physicians with those in Maryland and New York. Urinalysis, serum creatinine, serum cholesterol, and serum potassium tests are the most common laboratory tests ordered in all three settings but all tests except serum creatinine and potassium tests are less likely to be ordered in HCH settings, apparently because most HCH clinics do not have their own laboratories. One respondent noted, "Obtaining initial lab tests in our program requires sending a homeless hypertensive patient to our central health center." Resistance to treatment and absence of a fee source often preclude sending homeless hypertensives off-site for laboratory work. Routine tests are seen as needless complications or unaffordable luxuries.

Nondrug Therapies

We asked HCH physicians how frequently they recommended various nondrug therapies; the Maryland survey asked a somewhat different question that, nonetheless, provides interesting comparisons. In the Maryland study, the most important nondrug therapies, in descending order of importance, were sodium reduction, weight reduction, smoking reduction, increased exercise, and alcohol reduction. Among HCH physicians, alcohol reduction is by far the most important, followed at some distance by sodium reduction, weight reduction, and fat restriction. Alcohol abuse is strongly associated with blood pressure levels in all patient populations[9,10]; the greater importance accorded alcohol reduction by HCH physicians doubtlessly reflects the exceptional rate of alcohol abuse, especially among homeless men.

Dietary control of hypertension is obviously problematic in any population that depends on soup kitchens and bread lines for its sustenance. As one homeless hypertensive in New Orleans put it, "You gotta eat what they put on the plate." Since the prospects of dietary control for homeless patients are dim, they are accorded less importance in HCH clinics than elsewhere. The same can be said of other nondrug therapies often employed in conventional settings. Relaxation therapy is obviously pointless when maintaining the most basic necessities of life is a daily struggle. Smoking reduction may be equally pointless when cigarettes are the only remaining pleasure in life. The barriers to managing homeless hypertensives with nondrug therapies usually mean that antihypertensive drugs are the treatment of first *and* last resort.

Drug Therapies

Most HCH clinicians (45%) consider diuretics the treatment of choice, followed by calcium channel blockers (26%), beta blockers (12%), acetylcholinesterase inhibitors (11%), and "all others" (3%). Cost and availability of medications are more important factors in treating homeless hypertensives because the regimen is often life-long and therefore expensive. Nearly four fifths of HCH physicians cited cost as an important factor. Said one, "Newer and often more effective medicines can't be initiated because the homeless and the agencies that care for them can't afford them. The older medicines, with frequent dosing intervals and side effects, are the only ones we have available." Cost is not usually cited as a critical factor in HCH clinics that provide free medication.

Treatment and Compliance

A recent study of homeless hypertensives in New Orleans found that 59% had their blood pressures under control at least once in the course of treatment and 40% were in control at the last visit[2]; 60% of the domiciled hypertensives in a San Francisco study had normal blood pressure readings at their last visit.[11] Thus, effective treatment of homeless hypertensives is difficult but not entirely hopeless.[1,12,13]

One difficulty in treating the homeless is noncompliance, but this is also a problem for domiciled hypertensives.[14,15] Still, 91% of HCH physicians felt that compliance was more difficult for the homeless. Perceived barriers to compliance are summarized in Table 2. Alcohol abuse and availability of medication require no further comment. In domiciled populations, "The amount of social support a patient receives is strongly correlated with adherence to treatment" (p 355)[14]; among the homeless, estranged and disrupted social networks are common.[16] Further, approximately one homeless person in three is psychiatrically impaired.[17] Homeless patients frequently have trouble keeping appointments because they lack the routines of family and work, and many have no sense at all of day, month, or even year.

Strategies

How do HCH clinicians cope with the many difficulties summarized above? As one noted, with "Perseverance, patience, and creativity." To reduce confusion about medication, many HCH physicians prescribe once-daily dosages; to maintain contact, they schedule more frequent appointments and write prescriptions for shorter periods. Most HCH clinics also employ outreach workers to monitor clients in the streets. At least one HCH clinic has a "no-wait" drop-in station to provide immediate blood pressure readings. Many HCH facilities provide in-clinic medication storage. When possible, medication is dispensed in weather-resistant packages. At least three HCH clinics have experimented with clonidine transdermal patches, which eliminate the storage and daily-dosage problems altogether.[12,13]

TABLE 2—Relative Importance of Nine Factors in Impeding Compliance among Homeless Hypertensives: HCH Physician Assessments

Factor	Rated Importance[a]
Alcohol abuse	3.79
Unavailability of medication	3.76
Lack of social support network	3.42
Psychiatric impairment	3.40
Difficulty keeping appointments	3.34
Resistance to life-style changes	3.24
No place to store medication	3.10
Inadequate understanding of the effects of hypertension	3.08
Side effects of medication	2.63

Note. HCH = Health Care for the Homeless

[a] On a 1–4 scale where 1 = not important and 4 = very important.

One powerful incentive for hypertensives to maintain contact is that other needs can also be addressed at the HCH clinic. Most clinics have social workers, case managers, substance abuse counselors, and other service workers as well as medical personnel. Many of these clinics have evolved essentially into "one-stop" social service centers. Professional nutritionists can help with dietary management; 23% of the HCH clinics had a nutritionist on staff and an additional one third had access to nutritionists via referral.

The biggest barrier to treatment of homeless hypertensives, of course, is that their overall physical and mental well-being is threatened by much more pressing concerns than elevated blood pressure. Given their lack of food, clothing, shelter, and money, there is little concern among the homeless with preventive health measures or health maintenance.

Comment

Homelessness is a social condition with profound consequences for physical well-being, including the effective management of chronic physical disorders such as hypertension. The treatment of homeless hypertensives illustrates the folly inherent in strict biomedical models of disease and its alleviation. The range of treatments available, the course of the disorder, and the success of intervention are all powerfully affected by social factors.

It is an unfortunate commentary that we invest as much in providing health services for the homeless as we do in eradicating the basic causes of homelessness. People cannot be socially, mentally, or physically healthy without first having a stable and secure place to live. Health care clinics help homeless people lead more comfortable, less degrading lives, but they do little to reduce the number of homeless persons. Health care for the homeless, however creative, sensitive, and professionally motivated, cannot and does not address the more basic problems. The ultimate folly would be to become so proficient in dealing with the health consequences of homelessness that we lose sight of the fundamental problem of homelessness. ☐

References

1. Kellogg FR, Piantieri O, Conanan B, Doherty P, Vicic W, Brickner PW. Hypertension: A screening and treatment program for the homeless. In: Brickner PW et al., eds. *Health Care of Homeless People*. New York, NY: Springer-Verlag; 1985: 109–119.
2. Kinchen K. *The Prevalence, Management, and Consequences of Hypertension among the Homeless*. New Orleans, La: Tulane University; 1990. Thesis.
3. Institute of Medicine. *Homelessness, Health, and Human Needs*. Washington, DC: National Academy Press; 1988.
4. Wright JD, Weber E. *Homelessness and Health*. Washington, DC: McGraw-Hill; 1987.
5. Winick M. Nutritional and vitamin deficiency states. In: Brickner PW, Scharer LK, Conanan B, Elvy A, Savavese M, eds. *Health Care of Homeless People*. No. 1,5. New York, NY: Springer-Verlag; 1985.
6. Cloher T, Whelton P. Physician approach to the recognition and initial management of hypertension. *Arch Intern Med.* 1986; 146:529–533.
7. Thomson G, Alderman M, Wassertheil-Smoller S, Rafter J, Samet R. High blood pressure diagnosis and treatment: consensus recommendations vs. actual practice. *Am J Public Health*. 1981;71:413–416.
8. Joint National Committee. The 1988 report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med.* 1988; 148:1023–1038.
9. Kaplan N. *Clinical Hypertension*. 3rd and 4th eds. Baltimore, Md: Williams and Wilkins; 1986.
10. MacMahon S. Alcohol and hypertension: implications for prevention and treatment. *Ann Intern Med.* 1986;105:124–125.
11. Adamson T, Rodnick J, Guillion D. Family physicians and general internists: do they treat hypertensive patients differently? *J Fam Pract.* 1989;29:93–99.
12. Piantieri O, Vicic W, Byrd R, Brammer S, Michael M. Hypertension screening and treatment in the homeless. In: Brickner PW, Scharer LK, Conanan B, Savavese M, Scanlan BC, eds. *Under the Safety Net: The Health and Social Welfare of the Homeless in the United States*. New York, NY: WW Norton; 1990:250–262.
13. Nyamathi A, Shuler P. Factors affecting prescribed medication compliance of the urban homeless adult. *Nurse Pract.* 1989; 14:47–54.
14. Fedder D. Improving adherence to treatment regimens. *Md Med J.* 1988;37:354–356.
15. Klein L. Compliance and blood pressure control. *Hypertension*. 1988;11(suppl II):61–64.
16. Wright JD. *Address Unknown: The Homeless in America*. Hawthorne, NY: Aldine de Gruyter; 1989.
17. Wright JD. The mentally ill homeless: what is myth and what is fact? *Soc Prob.* 1988; 35(2):182–191.

Epidemiologic Studies Utilizing Surveys: Accounting for the Sampling Design

Edward L. Korn, PhD, and Barry I. Graubard, MA

ABSTRACT

Background. Since large-scale health surveys usually have complicated sampling schemes, there is often a question as to whether the sampling design must be considered in the analysis of the data. A recent disagreement concerning the analysis of a body iron stores-cancer association found in the first National Health and Nutrition Examination Survey and its follow-up is used to highlight the issues.

Methods. We explain and illustrate the importance of two aspects of the sampling design: clustering and weighting of observations. The body iron stores-cancer data are reanalyzed by utilizing or ignoring various aspects of the sampling design. Simple formulas are given to describe how using the sampling design of a survey in the analysis will affect the conclusions of that analysis.

Results. The different analyses of the body iron stores-cancer data lead to very different conclusions. Application of the simple formulas suggests that utilization of the sample clustering in the analysis is appropriate, but that a standard utilization of the sample weights leads to an uninformative analysis. The recommended analysis incorporates the sampling weights in a nonstandard way and the sample clustering in the standard way.

Conclusions. Which particular aspects of the sampling design to use in the analysis of complex survey data and how to use them depend on certain features of the design. We give some guidelines for when to use the sample clustering and sample weights in the analysis. (*Am J Public Health.* 1991;81:1166–1173)

Introduction

Many biomedical questions involving human populations cannot be answered by experimentation. We cannot ethically randomize patients to certain risk factors, e.g., exposure to asbestos. For factors we can manipulate, we may be unwilling to wait the required number of years or to follow the large number of individuals necessary for a meaningful analysis. Epidemiologic studies offer an alternative to randomized experiments that can avoid the major ethical problems and sometimes allow reduced sample sizes and earlier completion times.

Depending on the risk factors and outcomes, epidemiologic studies may still require large sample sizes. Large multipurpose health surveys can be a readily available source of data. An advantage of this type of data is that many risk factors can be examined. For example, consider the National and Hispanic Health and Nutrition Examination Surveys—NHANES I, II, III and HHANES—conducted in 1971–1975, 1976–1980, 1988–1994, and 1982–1984, respectively. Table 1 gives some examples of cross-sectional associations that have been studied using these surveys. Also, because these surveys were done at different times, one can examine cross-sectionally changes over time in various measured quantities (Table 1). In 1982–1984, a follow-up survey was conducted of NHANES I sampled individuals aged 25 to 74 years. This follow-up survey allows true longitudinal studies to be conducted (see Table 1 for examples). With diminishing financial resources, we believe that these types of surveys will be used increasingly to investigate biomedical and public health hypotheses.

A question that usually arises in the analysis of such survey data is whether the complex sampling design, which involves the clustering and weighting of observations, needs to be accounted for, or if it can be ignored by treating the sampled data as the population of interest. In some[5,7–12,15–20,22,23,25–36] of the above mentioned studies the sampling design was used in the analysis, while in others[1–4,6,13,14,21,24,37–53] it was not. The recent analysis by Stevens et al.[41] that ignored the sampling design in the analysis crystallizes the issue. Among other findings, Stevens et al.[41] found statistically significant differences in the mean total iron-binding capacity and mean transferrin saturation between men who developed cancer and men who did not, controlling for age and smoking status. Yip and Williamson[54] criticized this study because the sampling design was not used, and suggested that if it had been incorporated in the analysis, "it is doubtful that [these differences] would have remained 'statistically significant'." Stevens and colleagues replied, "It is important to take into account the probability sampling method used by NHANES when one is attempting to estimate the level of a variable in the US population at large. To test for differences between case patients and controls within the NHANES cohort, however, the methods we used are appropriate."[55] What is the correct approach to the analyses of these types of studies?

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This paper was submitted to the journal August 23, 1990, and accepted with revisions April 2, 1991.

TABLE 1—Examples of Studies Using Data from the National Health and Nutrition Examination Surveys

Outcome	References	Risk Factors/Subgroups
Cross-sectional associations		
Blood pressure	1–8	Nutrient intake; alcohol intake; blood lead levels; anthropometric measurements; race, sex, and age
Body weight	9–10	Smoking; alcohol
Anxiety	11	Aspects of diet
Periodontal disease	12	
Tooth decay	13	
General health	14	
Serum cholesterol	15	
Dietary supplements	16	
Blood iron levels	17	
Dietary pattern	18	
Constipation	19	
Glucose tolerance	20	Oral contraceptives; job characteristics
Cardiovascular disease	21–22	
Blood iron	23–24	Race, sex, and age
Blood lead	25	
Serum vitamin A	26	
Serum retinol	27	
Serum α-tocopherol	28	
Dietary intake	29	
Low birth weight	30	
Changes over time measured cross-sectionally		
Blood pressure	31	
Blood lead levels	32	
Anthropometric measurements	33	
Oral contraceptive use	34	
Incidence of childhood asthma	35 36	
Longitudinal studies		
Incidence of cancer	37–44	Alcohol intake; serum cholesterol; dietary fat; anthropometric measurements; blood iron; bowel function; physical activity; depression
Cardiovascular disease mortality	45	Cardiac size
Mortality from injuries	46	Alcohol consumption
Mortality (all causes)	47–50	Diabetes; race; antihypertensive drugs; self-rated health status
Depression	51	Physical activity
Hip fracture	52	Anthropometric measurements
Psychological well-being	53	Age

This article offers a brief tutorial about the sampling design and analysis of large household surveys. We examine whether the sampling design should be used when analyzing cross-sectional or longitudinal data derived from such surveys. We do not consider case-control studies, for which it is generally agreed that the sampling design must be considered in the analysis.[56–58] We consider conditions under which the sampling design can be safely ignored and the inefficiency resulting from utilizing the sampling design when it was unnecessary. The NHANES I and its follow-up are used as illustrations throughout, with special emphasis on the body iron stores-cancer association found by Stevens et al.[41]

Design of Complex Surveys: Cluster Sampling and Sample Weights

In this section the NHANES I design is used to describe the complex sampling design of household surveys. We stress the points of the design relevant to the issues discussed in this paper; other sources[59–61] provide further details. Although simple random samples are the easiest to analyze, they are impractical for large-scale national surveys for two reasons. First, no national registry of names to be sampled may exist. Second, even if a simple random sample of names could be obtained, it would be impractical to travel to, say, 20 000 locations scattered throughout the United States to interview the sampled individuals. Multistage survey designs, on the other hand, enable sampling frames to be developed for a relatively small number of areas and limit the number of locations to which interviewers must travel. For example, in the first stage of NHANES I sampling, the mainland United States was divided into approximately 1900 geographic areas or primary sampling units (PSUs), each one consisting of a standard metropolitan statistical area or, at most, three contiguous counties. These PSUs were grouped into 40 strata from each of which one or two PSUs were sampled; some very large PSUs were sampled with certainty. In each of these sampled PSUs, successive stages of sampling involved census enumeration districts (approximately 300 households), segments (approximately 8 contiguous housing units), and sampled individuals.

At first glance accounting for this type of multistage clustering in the analysis of survey data seems a formidable task. Fortunately, approximate analytic methods have been developed that require only the knowledge of the stratum and PSU of each individual.[62] In practice, computer data files from large surveys contain this information so that it can be readily accessed by statistical computer packages. For NHANES I, the design can be approximated[60] for the statistical analysis as a selection of 70 PSUs, two from each of 35 strata.

For complex surveys, each sampled individual with data has a sample weight associated with his or her data. The sample weight is the number of individuals in the target population that the sampled individual represents. The sample weight may be derived as the product of three components. The first comes from the fact that surveys frequently over-sample certain groups in the population (base weight). For example, in NHANES I, people living in poverty census enumeration districts were sampled as much as eight times more often than people living elsewhere. Additionally, persons aged 65 years or older were sampled twice as often as women ages 20–44, who in turn were sampled twice as often as other adults.

TABLE 2—Adjusted Mean Values[a] for Total Iron-Binding Capacity for Subjects in Whom Cancer Developed and Those Who Remained Cancer-free

	Clustering Used?	Sample Weights Used?	Cancer[b]	No Cancer[c]	Difference Mean±SE	Difference 95% CI	P
				Men			
A[d]	No	No	61.30	62.78	−1.48±0.60	−2.65 to −0.31	.013
B	No	Yes	62.65	63.57	−0.92±1.06	−2.99 to 1.15	.38
C[e]	Yes	No	61.30	62.78	−1.48±0.54	−2.58 to −0.38	.010
D[e]	Yes	Yes	62.65	63.57	−0.92±0.86	−2.67 to 0.83	.29
E	Yes	Partly[f]	62.07	63.51	−1.44±0.53	−2.51 to −0.37	.010
				Women			
A[d]	No	No	66.32	66.40	−0.08±0.79	−1.62 to 1.46	.92
B	No	Yes	65.60	66.32	−0.73±1.18	−3.04 to 1.58	.54
C[e]	Yes	No	66.32	66.40	−0.08±0.69	−1.48 to 1.33	.91
D[e]	Yes	Yes	65.60	66.32	−0.73±1.29	−3.34 to 1.88	.58
E	Yes	Partly[f]	66.20	66.35	−0.16±0.60	−1.38 to 1.06	.82

Note. SE = standard error; CI = confidence interval.

[a] Means are adjusted for age and smoking status. For the analysis labeled E, means are also adjusted for race (White vs non-White), family income (<$3000, $3000–$6999, $7000–$9999, $10 000–$14 999, ≥$15 000), poverty vs nonpoverty census enumeration district, and senior status (age ≥65 or <65). For the 3.8% of the sample whose family income was unavailable, income was imputed from education of the head of household in the same manner used in the construction of the sample weights.

[b] For men, N = 242; for women, N = 203.

[c] For men, N = 3116; for women, N = 5165.

[d] Analyses labeled A are from Stevens et al.[41] Adjusted means for cancer and no cancer groups are slightly different here because their analysis implicitly assumed that all four smoking categories were equally likely. Analyses here use observed proportions in each smoking category (analyses A and B) or estimated population proportions in each category (analyses C, D, and E).

[e] Mean estimates for analysis C and D are the same as estimates for analysis A and B, respectively.

[f] Analyses labeled E are unweighted regressions with means adjusted for many of the variables used in defining sample weights (see footnote a).

Finally, in a small number of segments there were more households encountered by the interviewers than expected. These households were subsampled at rates of one half to one quarter.

The second component of the sample weight is an adjustment for nonresponse, including both the inability to locate sampled individuals and their refusal to participate. Although there is no ideal approach to adjust for nonresponse, adjustment using weights is as follows. The population is divided into groups so that the probability of nonresponse is thought to be roughly similar for all individuals within a group. The data from respondents is then weighted upwards depending on the nonresponse rate of their corresponding group. For example, in NHANES I the nonresponse groups were based on five family income groups within each PSU.

The third component of the sample weight is an adjustment so that the sum of the weights for a given sex, race, and age agree with known population figures (poststratification adjustment). Finally, because not all sampled persons in NHANES I had their body iron stores measured, a special set of weights was derived for use in the analysis of these data.

Reanalysis of the Body Iron Stores-Cancer Association

In this section we follow the analyses given in Stevens et al.[41] with the exception of using the sample clustering and/or the sample weights. We demonstrate how the conclusions of the analysis change when these two aspects of the sampling design are incorporated. We restrict attention to the associations described by Stevens et al.[41] of total iron-binding capacity (mol/L) and transferrin saturation (%) with the development of cancer. Briefly, the data to be analyzed are from 3358 men and 5368 women who (1) had their total iron-binding capacity measured in NHANES I in 1971–1975, (2) had their cancer status determined in the follow-up survey in 1981–1984, and (3) were alive and cancer-free for at least 4 years after their biochemical measurements. These sample sizes are slightly different from those of Stevens et al.[41] because the follow-up public use data files have been revised since their analyses.

Although there are many ways to analyze this type of follow-up data, we follow the simplest analysis given by Stevens et al.,[41] which compares the mean biochemical values in those respondents who developed cancer and those who did not, adjusted by linear regression for age (years) and smoking status (current smoker, former smoker, never a smoker, and unknown). Tables 2 and 3 contain the results of five different analyses of these same associations, labeled A through E.

The A analyses repeat the Stevens et al.[41] analyses. We conclude from the A analyses that for both variables there is a small but statistically significant association for men and no association for women.

The B analyses use the sample weights of the survey but not the clustering. A dramatic effect of using the weights is the increase in the standard errors (SEs), the most extreme case being the change from 0.79 to 2.53 for the SE of the difference in adjusted means for the women's transferrin saturation. The correct interpretation for this particular weighted analysis is not that there is no association between transferrin saturation and the development of cancer in women. Instead, it is that because the SE and confidence interval (CI) are so large, the data on the association are uninformative.

The C and D analyses use the sample clustering, without and with the sample weights, respectively. Only the SEs, not the means, are possibly affected by use of the clustering. For these particular analyses, it turns out that the standard errors are not much affected. The results and conclusions, therefore, are very similar to the unclustered A and B analyses, respectively.

We recommend the E analyses for several reasons. They are unweighted analyses that use the sample clustering, but for which the adjusted means have been further adjusted by linear regression for many of the variables used in defining the sample weights (see footnote a of Table 2). The results and conclusions of the E analyses turn out to be very similar to the unweighted unclustered A analyses. This happens to be true even though these analyses do not ignore the sampling weights and clustering.

The computer programs REG[63] and SURREGR[64] were used to perform the analyses A and B–E, respectively. Because 3 of the PSUs have no total iron-binding capacity data available, for the C, D, and E analyses the strata containing these PSUs are pooled with neighboring strata to yield a design approximated by 67 PSUs selected from 32 strata. The analyses A–E give an idea of the different conclusions that are possible when using various aspects of the sample design in the analysis. In the sections below, we give some rules of thumb for choosing the most appropriate analyses based on the sample survey design alone.

Should Sample Clustering Be Used in Analysis?

Sample clustering can affect the variability (SEs) of estimates of associations. First, a simple but extreme example will show this. Consider a simple random sample of 1000 households, where disease and risk factor status are obtained from each member of the household. Suppose that if one member of a household has the risk factor, then all members of the household have it, and if one member of the household has the disease, then all have it. The appropriate sample size for the analysis of this sampled data is 1000, not the number of individuals sampled. Thus, for example, if 4000 individuals had been sampled in the 1000 households, and the sample (household) clustering was ignored in the analysis, then SEs would be too small by a factor of 2 $(= \sqrt{4000/1000})$, because SEs are inversely proportional to sample sizes. Admittedly, this is an extreme example to make the point; in general the effect of clustering depends on both the cluster size and the intraclass correlation.[62]

TABLE 3—Adjusted Mean Values[a] for Transferrin Saturation (%) for Subjects in Whom Cancer Developed and Those Who Remained Cancer-free

	Clustering Used?	Sample Weights Used?	Cancer[b]	No Cancer[c]	Difference		
					Mean±SE	95% CI	*P*
				Men			
A[d]	No	No	33.15	30.73	2.42±0.78	0.89–3.94	.002
B	No	Yes	32.55	30.43	2.12±1.48	−0.78–5.03	.15
C[e]	Yes	No	33.15	30.73	2.42±0.86	0.68–4.16	.008
D[e]	Yes	Yes	32.55	30.43	2.12±1.22	−0.35–4.59	.09
E	Yes	Partly[f]	32.92	30.48	2.44±0.85	0.71–4.17	.007
				Women			
A[d]	No	No	27.98	27.17	0.81±0.79	−0.73–2.36	.30
B	No	Yes	31.37	27.74	3.63±2.53	−1.32–8.58	.15
C[e]	Yes	No	27.98	27.17	0.81±0.76	−0.72–2.35	.29
D[e]	Yes	Yes	31.37-	27.74	3.63±2.42	−1.28–8.53	.14
E	Yes	Partly[f]	28.36	27.57	0.78±0.74	−0.72–2.28	.30

Note: SE = standard error; CI = confidence interval.

[a] Means are adjusted for age and smoking status. For the analysis labeled E, means are also adjusted for race (White vs non-White), family income (under \$3000, \$3000–\$6999, \$7000–\$9999, \$10 000–\$14 999, ≥\$15 000), poverty vs nonpoverty census enumeration district, and senior status (age ≥65 or <65). For the 3.8% of the sample whose family income was unavailable, income was imputed from education of the head of household in the same manner used in the construction of the sample weights.

[b] For men, N = 232; for women, N = 197.

[c] For men, N = 3056; for women, N = 5073.

[d] Analyses labeled A are from Stevens et al.[41] Adjusted means for cancer and no cancer groups are slightly different here because their analysis implicitly assumed that all four smoking categories were equally likely. Analyses here use observed proportions in each smoking category (analyses A and B) or estimated population proportions in each category (analyses C, D, and E).

[e] Mean estimates for analysis C and D are the same as the estimates for analysis A and B, respectively.

[f] Analyses labeled E are unweighted regressions with means adjusted for many of the variables used in defining sample weights (see footnote a).

In many realistic situations, the "disease" may be a continuous outcome (e.g., blood pressure) and the risk factor may also be a continuous variable (e.g., dietary calcium intake). Additionally, the risk factor-disease association of interest may be the one in which certain covariates are controlled for, e.g., sex and age. A general sufficient condition for ignoring the clustering in the analysis is the distribution of the outcome for given levels of the risk factor and covariates does not depend on which cluster the individual is in. Given the complicated multistage sampling in a survey such as NHANES I, it may not be obvious whether this condition is satisfied or not.

Since adjusting for the sample clustering will on average increase the SEs, its use in the analysis can be thought of as a conservative procedure. What is lost by using the clustering in the analysis when it was unnecessary? Unnecessary clustering leads to an inefficient analysis, that is, an analysis with CIs wider than necessary and with less statistical power for rejecting the null hypothesis. An easy way to express this inefficiency is in terms of relative sample sizes. For example, an analysis that is 20% inefficient with respect to another analysis means that a sample size of 1000 in the inefficient analysis is equivalent to a sample size of 800 in the more efficient analysis. A key number for estimating the inefficiency of using clustering when unnecessary is the degrees of freedom, d, available for the estimation of SEs. The d is usually taken to be the number of sampled PSUs minus the number of strata. For example, in the NHANES I analysis of body iron stores and cancer, d = 35 = 67 − 32.

The approximate inefficiency in doing an analysis that uses the sample clustering when unnecessary is:[65]

$$\text{Inefficiency} = 1 - (z^{1-\alpha/2}/t_d^{\,1-\alpha/2})^2$$

where $z^{1-\alpha/2}$ and $t_d^{\,1-\alpha/2}$ are the $1 - \alpha/2$ percentiles of a normal distribution and a t-distribution with d degrees of freedom, respectively, and α is the significance level for testing or one minus the confidence level for CIs. For example, for the analyses of NHANES I given in the previous section in which we used 95% CIs for the mean differences, suppose we are also interested in hypothesis testing at the $\alpha = .05$ level. Because d = 35, any standard set of statistical tables gives $z^{0.975} = 1.96$ and $t_{35}^{\,.975} = 2.03$. The inefficiency equals 6.8%, an inefficiency so small that one should use the clustering in the analyses. If the clustering is important, it must be used so that the variability of means is properly estimated; if the clustering is unimportant, little is lost by using it unnecessarily. The similar magnitude of the SEs for analyses A and C in Tables 2 and 3 suggest that, in fact, clustering was not important for these data.

In general, the inefficiency of using the sample clustering when unnecessary becomes greater with decreasing degrees of freedom. For example, the design of HHANES can be approximated[66] by sampling 16 PSUs from 8 strata. For this design, the inefficiency is 28% when the clustering is unnecessary (using d = 8 in the above formula), making the decision of whether or not to use the clustering in the analysis more difficult than with NHANES I. These inefficiency calculations are meaningful whenever a single disease-risk factor association is being examined. For simultaneous analysis of many disease-risk factor associations or other multivariate analyses, the inefficiency of using the clustering when unnecessary can increase.[67]

Should Sampling Weights Be Used in Analysis?

It is well known that the unweighted mean of a sample that is not a simple random sample can lead to a biased estimate

TABLE 4—Percentiles of Sample Weights for Individuals Analyzed

Sex	Minimum	5%	25%	Median	75%	95%	Maximum
Men (n = 3358)	477	876	3614	8170	19 504	31 556	135 824
Women (n = 5368)	611	114[illegible]	3162	6684	11 119	24 090	186 062

of the population mean, whereas a weighted mean provides an approximately unbiased estimate. It is not as obvious that an unweighted analysis of a disease-risk factor association can also lead to the wrong conclusions. A simple numerical example given in the Figure shows this to be true. The expected numbers of sampled individuals are given in the Figure for two sampling schemes for the whole population was well as within two subgroups defined by area of residence. On the left, a study using a simple random sample of 10 000 individuals would show on average the correct result that an association exists between the disease and the risk factor: The probability of having the disease is 5.9% without the risk factor and 12.2% with the risk factor, for a relative risk of approximately 2. On the right, a study of the same population that samples nonurban area residents at nine times the rate of urban area residents would on average sample the same number of individuals from each type of area. Unweighted analysis of data from this probability sample suggests incorrectly that there is no association between the disease and the risk factor. A weighted analysis of the data from this probability sample would on average yield the correct association:

$$2.06 = \frac{(9\times245 + 655)/[(9\times245 + 655) + (9\times4655 + 3445)]}{(9\times15 + 85)/[(9\times15 + 85) + (9\times85 + 815)]}$$

A weighted analysis will provide an approximately unbiased estimate of the population association, whereas an analysis ignoring the weights, in general, will not. A general sufficient condition for when the sampling weights can be ignored, however, is as follows: The distribution of the outcome (disease) for given levels of the risk factor and covariates does not depend on any variables used in the sampling design or used to adjust for nonresponse.

Because it may be difficult to verify this sufficient condition, what is the harm in always using the sample weights in the analysis? As with the use of clustering when unnecessary, the use of sampling weights when unnecessary leads to an inefficient analysis. This inefficiency can be defined as

$$1 - (\mathrm{SE}_{\mathrm{unwtd}}/\mathrm{SE}_{\mathrm{wtd}})^2$$

where $\mathrm{SE}_{\mathrm{unwtd}}$ and $\mathrm{SE}_{\mathrm{wtd}}$ are the unweighted and weighted SEs of the mean difference. For the linear regression models used in the analyses here, the calculation of the efficiency depends on the distribution of the sample weights and independent variables (see DuMouchel and Duncan[68] for the exact formulas). An approximation is given by the following formula:

$$\text{Inefficiency} = 1 - (w_1 + w_2 + \ldots + w_N)^2/[N(w_1^2 + w_2^2 + \ldots + w_N^2)]$$

where $w_1, w_2, \ldots, w_N$ are the sample weights for the N sampled individuals. The inefficiency increases when the sample weights are more variable, and is zero if all the sample weights are identical.

Table 4 presents selected percentiles of the distribution of sample weights for the individuals analyzed here. The range of the weights is very large, with the largest weight 285 (300) times greater than the smallest weight for the men (women). The inefficiencies of using the sample weights when unnecessary are calculated to be 47% and 48% for men and women, respectively. Considering that the unweighted SEs are not insubstantial compared to clinically meaningful treatment differences, these inefficiencies are too large for us to assert that little is lost by using the weights when unnecessary.

There is an additional consideration when using the sample weights in the analysis. If the sample weights do matter, they can make the analysis even less efficient than described above. For example, consider the analysis of the transferrin saturation in women given in Table 3. Comparing analysis A to analysis B, the SE has increased by a factor of 3.2 = 2.53/0.79, suggesting an inefficiency of 90%. A closer look at the data for this analysis is instructive. Out of the 197 women who developed cancer, the woman with the highest transferrin saturation (68.4%) also had the third largest sample weight (103 042) out of all 5368 women. If this woman's sample weight had had a median value of 6684, then the mean difference in the weighted analysis B would have been 1.35 ± 1.16 instead of 3.63 ± 2.53.

Since the inefficiency of performing a weighted analysis is unacceptably high, what do we recommend? One approach is to use an unweighted analysis but to control for the variables utilized in determining the sample weights. In the present context, the E analyses in Tables 2 and 3 compute the means adjusted for many of these variables. One could argue[69] that because these additional variables (family income, race, etc.) are highly unlikely to be on the causal pathway between body iron stores and the development of cancer, it would be useful to control for these variables even if we had a simple random sample. We view this not as the perfect solution, but as a compromise that avoids the inefficiency of a weighted analysis. As it happens, the results of the E analyses are remarkably similar to the unweighted, unclustered A analyses, but, we can now have more confidence in these results. However, for other data sets and other associations this may not be the case.

Discussion

The impact of sampling design on the analysis of complex survey data should always be considered. Which particular aspects of the design to use in the analysis, however, is a subtler question. Classical survey analysts recommend using both the clustering and the sample weights in the analysis.[70,71] Although this is a philosophically defensible position, we have seen above that it can lead to inferences that make the whole study worthless. On the other hand, the position of Stevens et al.[55] that the sampling design can be ignored when addressing these types of questions is tenuous unless one is willing to make strong assumptions. We have attempted in this article to give simple ways of weighing the costs and benefits of accounting for the sampling design in the analysis. For specific recommendations concerning sample clustering and sample weights, we offer the following rules of thumb.

(1) If the number of sampled PSUs minus the number of strata is greater than or equal to 20 (corresponding to an inef-

SIMPLE RANDOM SAMPLING

(N= 10000 sampled from entire population)

Totals		Disease		Proportion
		-	+	with Disease
Risk	-	9068	572	5.9%
Factor	+	316	44	12.2%
		Total 10000		

Subgroup 1 (Urban Area)		Disease		
		-	+	
Risk	-	8379	441	5.0%
Factor	+	153	27	15.0%
		Total 9000		

Subgroup 2 (Non-Urban Area)		Disease		
		-	+	
Risk	-	689	131	16.0%
Factor	+	163	17	9.4%
		Total 1000		

PROBABILITY SAMPLING

(N= 5000 sampled from each subgroup)

Totals		Disease		Proportion
		-	+	with Disease
Risk	-	8100	900	10.0%
Factor	+	900	100	10.0%
		Total 10000		

Subgroup 1 (Urban Area)		Disease		
		-	+	
Risk	-	4655	245	5.0%
Factor	+	85	15	15.0%
		Total 5000		

Subgroup 2 (Non-Urban Area)		Disease		
		-	+	
Risk	-	3445	655	16.0%
Factor	+	815	85	9.4%
		Total 5000		

FIGURE—unweighted analysis of data from a probability sample can lead to incorrect conclusions

ficiency of roughly 10%), then use the clustering in the analysis. Otherwise, more advanced statistical methods[72] to account for the clustering can be applied to roughly approximate the SEs of the parameters of interest.

(2) Calculate the approximate inefficiency of including the sample weights using the simple formula given in the previous section. If this inefficiency is less than 10%, then use the sample weights in the analysis. If this inefficiency is greater than 10%, then consider the effect this inefficiency will have on the SEs compared to an expected clinically meaningful treatment difference. If the effect is not unacceptably large, use the sample weights in the analysis. Otherwise, use an "unweighted" analysis that controls for the variables relating to the design and nonresponse adjustments in the analysis.

We end with two suggestions for the designers and producers of large-scale health surveys that will be used by other investigators. If our recommendations are followed, one will sometimes need to use variables relating to the design and nonresponse adjustments of the survey. Our first suggestion is that all these variables be documented and included in public use data files.

Our second recommendation concerns the design of the surveys themselves. As we have discussed, a small number of sampled PSUs or individuals with extremely high sample weights can make the use of the sample design in the analysis very inefficient. Given the large number of secondary analyses that are performed using these survey data, we suggest that policymakers provide sufficient resources so that the surveys can be designed for efficient secondary analyses using the design. □

Acknowledgments

We are indebted to L.S. Freedman, S.B. Green, D. Hitchcock, M.G. Kovar, J.L. Mills, B.H. Patterson, and R.G. Stevens for their helpful comments.

References

1. Blair D, Habicht JP, Sims EAH, Sylwester D, Abraham S. Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. *Am J Epidemiol.* 1984;119:526–540.
2. Frisancho AR, Leonard WR, Bollettino LA. Blood pressure in blacks and whites and its relationship to dietary sodium and potassium intake. *J Chron Dis.* 1984;37:515–519.
3. Stanton JL, Braitman LE, Riley AM, Khoo CS, Smith JL. Demographic, dietary, life style, and anthropometric correlates of blood pressure. *Hypertension.* 1982;4(suppl III):135–142.
4. Gruchow HW, Sobocinski KA, Barboriak JJ. Alcohol, nutrient intake, and hypertension in US adults. *JAMA.* 1985;253:1567–1570.
5. Harlan WR, Hull AL, Schmouder RL, Landis JR, Thompson FE, Larkin FA. Blood pressure and nutrition in adults. *Am J Epidemiol.* 1984;120:17–28.
6. McCarron DA, Morris CD, Henry HJ, Stanton JL. Blood pressure and nutrient intake in the United States. *Science.* 1984;224:1392–1398.
7. Sempos C, Cooper R, Kovar MG, Johnson C, Drizd T, Yetley E: Dietary calcium and blood pressure in NHANES I and II. *Hypertension.* 1986;8:1067–1074.
8. Harlan WR, Landis JR, Schmouder RL, Goldstein NG, Harlan LC. Blood lead and blood pressure. *JAMA.* 1985;253:530–534.
9. Albanes D, Jones Y, Micozzi MS, Mattson ME. Associations between smoking and body weight in the US population: analysis of NHANES II. *Am J Public Health.* 1987;77:439–444.
10. Williamson DF, Forman MR, Binkin NJ, Gentry EM, Remington PL, Trowbridge FL. Alcohol and body weight in United States adults. *Am J Public Health.* 1987;77:1324–1330.
11. Eaton WW, McLeod J. Consumption of coffee or tea and symptoms of anxiety. *Am J Public Health.* 1984;74:66–68.
12. Ismail AI, Burt BA, Eklund SA. Relation between ascorbic acid intake and periodontal disease in the United States. *J Am Dent Assoc.* 1983;107:927–931.
13. Ismail AI, Burt BA, Eklund SA. The cariogenicity of soft drinks in the United States. *J Am Dent Assoc.* 1984;109:241–245.
14. Schwerin HS, Stanton JL, Riley AM, et al. Food eating patterns and health: a reexamination of the Ten-State and HANES I surveys. *Am J Clin Nutr.* 1981;34:568–580.
15. Kovar MG, Fulwood R, Feinleib M. Coffee studies. *N Engl J Med.* 1983;309 (To the Editor):1249.
16. Koplan JP, Annest JL, Layde PM, Rubin GL. Nutrient intake and supplementation in the United States (NHANES II). *Am J Public Health.* 1986;76:287–289.
17. Looker AC, Sempos CT, Johnson CL, Yetley EA. Comparison of dietary intakes and iron status of vitamin-mineral supplement users and nonusers, aged 1–19 years. *Am J Clin Nutr.* 1987;46:665–672.
18. Schectman G, McKinney WP, Pluess J, Hoffman RG. Dietary intake of Americans

reporting adherence to a low cholesterol diet (NHANES II). *Am J Public Health.* 1990;80:698–703.

19. Sandler RS, Jordan MC, Shelton BJ. Demographic and dietary determinants of constipation in the US population. *Am J Public Health.* 1990;80:185–189.
20. Russell-Briefel R, Ezzati TM, Perlman JA, Murphy RS. Impaired glucose tolerance in women using oral contraceptives: United States, 1976–80. *J Chronic Dis.* 1987;40:3–11.
21. Russell-Briefel R, Ezzati TM, Fulwood R, Perlman JA, Murphy RS. Cardiovascular risk status and oral contraceptive use: United States, 1976–80. *Prev Med.* 1986;15:352–362.
22. Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). *Am J Public Health.* 1988;78:910–918.
23. Dallman PR, Yip R, Johnson C. Prevalence and causes of anemia in the United States, 1976 to 1980. *Am J Clin Nutr.* 1984;39:437–445.
24. Meyers LD, Habicht JP, Johnson CL. Components of the difference in hemoglobin concentrations in blood between black and white women in the United States. *Am J Epidemiol.* 1979;109:539–549.
25. Mahaffey KR, Annest JL, Roberts J, Murphy RS. National estimates of blood lead levels: United States, 1976–80. *N Engl J Med* 1982;307:573–579.
26. Looker AC, Johnson CL, Woteki CE, Yetley EA, Underwood BA. Ethnic and racial differences in serum vitamin A levels of children aged 4–11 years. *Am J Clin Nutr.* 1988;47:247–252.
27. Looker AC, Johnson CL, Underwood BA. Serum retinol levels of persons aged 4–74 years from three Hispanic groups. *Am J Clin Nutr.* 1988;48:1490–1496.
28. Looker A, Underwood B, Wiley J, Fulwood R, Sempos C. Serum α-tocopherol levels of Mexican Americans, Cubans, and Puerto Ricans aged 4–74 y. *Am J Clin Nutr.* 1989;50:491–496.
29. Block G, Rosenberger WF, Patterson BH. Calories, fat and cholesterol: intake patterns in the US population by race, sex, and age. *Am J Public Health.* 1988;78:1150–1155.
30. Scribner R, Dwyer JH. Acculturation and low birthweight among Latinos in the Hispanic HANES. *Am J Public Health.* 1989;79:1263–1267.
31. Dannenberg AL, Drizd T, Horan MJ, Haynes SG, Leaverton PE. Progress in the battle against hypertension: blood pressure trends in the United States from 1960 to 1980. *Hypertension.* 1987;10:226–233.
32. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. *N Engl J Med.* 1983;308:1373–1377.
33. Flegal KM, Harlan WR, Landis JR. Secular trends in body mass index and skinfold thickness with socioeconomic factors in young adult women. *Am J Clin Nutr.* 1988;48:535–543.
34. Harlan WR, Landis JR, Flegal KM, Davis CS, Miller ME. Secular trends in body mass in the United States, 1960–80. *Am J Epidemiol.* 1988;128:1065–1074.
35. Russell-Briefel R, Ezzati T, Perlman J. Prevalence and trends in oral contraceptive use in premenopausal females ages 12–54 years, United States, 1971–80. *Am J Public Health.* 1985;75:1173–1176.
36. Gergen [illegible] Mullally DI, Evans R. National survey of prevalence of asthma among children [illegible] the United States, 1976–80. *Pediatrics.* [illegible]988;81:1–7.
37. Schatzkin A, Jones DY, Hoover RN, et al. Alcohol consumption and breast cancer in the epidemiologic follow-up study of the first National Health and Nutrition Examination Survey. *N Engl J Med.* 1987;316:1169–1173.
38. Schatzkin A, Taylor PR, Carter CL, et al. Serum cholesterol and cancer in the NHANES I Epidemiologic Follow-up Study. *Lancet II.* 1987;298–301.
39. Jones DY, Schatzkin A, Green SB, et al. Dietary fat and breast cancer in the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. *JNCI.* 1987;79:465–471.
40. Swanson CA, Jones DY, Schatzkin A, Brinton LA, Ziegler RG. Breast cancer risk assessed by anthropometry in the NHANES I Epidemiological Follow-up Study. *Cancer Res.* 1988;48:5363–5367.
41. Stevens RG, Jones DY, Micozzi MS, Taylor PR. Body iron stores and the risk of cancer. *N Engl J Med.* 1988;319:1047–1052.
42. Micozzi MS, Carter CL, Albanes D, Taylor PR, Licitra LM. Bowel function and breast cancer in US women. *Am J Public Health.* 1989;79:73–75.
43. Albanes D, Blair A, Taylor PR. Physical activity and risk of cancer in the NHANES I population. *Am J Public Health.* 1989;79:744–750.
44. Zonderman AB, Costa PT, McCrae RR. Depression as a risk for cancer morbidity and mortality in nationally representative sample. *JAMA.* 1989;262:1191–1195.
45. Rautaharju PM, LaCroix AZ, Savage DD, et al. Electrocardiographic estimate of left ventricular mass versus radiographic cardiac size and the risk of cardiovascular disease mortality in the Epidemiologic Follow-up Study of the first National Health and Nutrition Examination Survey. *Am J Cardiol.* 1988;62:59–66.
46. Anda RF, Williamson DF, Remington PL. Alcohol and fatal injuries among US adults. *JAMA.* 1988;260:2529–2532.
47. Kleinman JC, Donahue RP, Harris MI, Finucane FF, Madans JH, Brock DW. Mortality among diabetics in a national sample. *Am J Epidemiol.* 1988;128:389–401.
48. Otten MW, Teutsch SM, Williamson DF, Marks JS. The effect of known risk factors on the excess mortality of black adults in the United States. *JAMA.* 1990;263:845–850.
49. Havlik RJ, La Croix AZ, Kleinman JC, Ingram DD, Harris T, Cornoni-Huntley J. Antihypertensive drug therapy and survival by treatment status in a national survey. *Hypertension.* 1989;13:I28–I32.
50. Idler EL, Angel RJ. Self-rated health and mortality in the NHANES-I Epidemiologic Follow-up Study. *Am J Public Health.* 1990;80:446–452.
51. Farmer ME, Locke BZ, Moscicki EK, Dannenberg AL, Larson DB, Radloff LS. Physical activity and depressive symptoms: the NHANES I Epidemiologic Follow-up Study. *Am J Epidemiol.* 1988;128:1340–1351.
52. Farmer ME, Harris T, Madans JH, Wallace RB, Cornoni-Huntley J, White LR. Anthropometric indicators and hip fracture: the NHANES I Epidemiologic Follow-up Study. *J Am Geriatr Soc.* 1989;37:9–16.
53. Costa PT, Zonderman AB, McCrae RR, Cornoni-Huntley J, Locke BZ, Barbano HE. Longitudinal analyses of psychological well-being in a national sample: stability of mean levels. *J Gerontol.* 1987;42:50–55.
54. Yip R, Williamson DF. Body iron stores and risk of cancer. *N Engl J Med.* 1989;320 (To the Editor):1012.
55. Stevens RG, Jones DY, Micozzi MS, Taylor PR. Body iron stores and risk of cancer. *N Engl J Med.* 1989;320 (To the Editor):1014.
56. Scott AJ, Wild CJ. Fitting logistic models under case-control or choice based sampling. *J R Stat Soc B.* 1986;48:170–182.
57. Cain KC, Breslow NE. Logistic regression analysis and efficient design for two stage studies. *Am J Epidemiol.* 1988;126:1198–1206.
58. Graubard BI, Fears TR, Gail MH. Effects of cluster sampling on epidemiologic analysis in population-based case-control studies. *Biometrics.* 1989;45:1053–1071.
59. Miller HW. Plan and operation of the Health Nutrition Examination Survey. *Vital Health Stat.* 1985;1(10a). Hyattsville, Md: National Center for Health Statistics.
60. Landis JR, Lepkowski JM, Eklund SA, Stehouwer SA. A statistical methodology for analyzing data from a complex survey: the First National Health and Nutrition Examination Survey. *Vital Health Stat.* 1982;2(92). Hyattsville, Md: National Center for Health Statistics.
61. Cohen BB, Barbano HE, Cox CS, et al. Plan and operation of the NHANES I Epidemiologic Follow-up Study: 1982–84. *Vital Health Stat.* 1987;1(22). Hyattsville, Md: National Center for Health Statistics.
62. Wolter KM. *Introduction to Variance Estimation.* New York, NY: Springer-Verlag; 1985.
63. SAS Institute Inc. *SAS User's Guide: Statistics, Version 5 edition.* Cary, NC: SAS Institute; 1985.
64. Shah BV. SURREGR: *Standard Errors of Regression Coefficients from Sample Survey Data.* Research Triangle Park, NC: Research Triangle Institute; 1982.
65. Cornfield J. Randomization by group: a formal analysis. *Am J Epidemiol.* 1978;108:100–102.
66. Russell-Briefel R, Dresser CM, Ezzati TM, et al. Plan and operation of the Hispanic Health and Nutrition Examination Survey 1982–1984. *Vital Health Stat.* 1985;1(19). Hyattsville, Md: National Center for Health Statistics.
67. Korn EL, Graubard BI. Simultaneous testing of regression coefficients with complex

survey data: use of Bonferroni t-statistics. *The American Statistician.* 1990;44:270–276.

68. DuMouchel WH, Duncan GJ. Using sample survey weights in multiple regression analysis of stratified samples. *J Am Stat Assoc.* 1983;78:535–543.
69. Korn EL, Graubard BI. Examining neighborhood confounding in a survey: an example using the National Health and Nutrition Examination Survey II. *Stat Med.* 1988;7:1087–1098.
70. Kish L, Frankel MR. Inference from complex samples. *J R Stat Soc* Ser. B. 1974;36:1–37.
71. Hansen MH, Madow WG, Tepping BJ. An evaluation of model-dependent and probability-sampling inferences in sample surveys. *J Am Stat Assoc.* 1983;78:776–793.
72. Pfeffermann D, LaVange L. Regression models for stratified multi-stage cluster samples. In: Skinner CJ, Holt D, Smith TMF, eds. *Analysis of Complex Surveys.* New York, NY: John Wiley & Sons; 1989.

Drug with Potential for Treating the Neuropsychological Effects of HIV Infection to Be Tested

The National Institute of Mental Health (NIMH) today announced the beginning of a clinical study of the effectiveness of Peptide T, a drug of potential value in treating the neuropsychological effects of HIV infection and AIDS.

"This is the first study designed to answer questions about the effectiveness of Peptide T by comparing the compound with a placebo in a controlled study design," said Alan I. Leshner, PhD, NIMH acting director.

The first patient started treatment with Peptide T in mid-March. The study, which is jointly sponsored by NIMH and the National Institute of Allergy and Infectious Diseases, is being conducted at the University of Southern California in Los Angeles.

The human immunodeficiency virus is believed to cause problems of concentration and memory in some persons with AIDS and AIDS-related complex by directly affecting the brain.

Peter N.R. Heseltine, MD, the principal investigator, said "in earlier studies, people with these problems have shown improvement in tests of their mental function when they were treated with Peptide T. The purpose of this new study is to discover if Peptide T is safely able to reduce these HIV-caused problems of concentration and memory and if it is safe and effective when used with the antiretroviral AZT (zidovudine)."

Ellen Stover, PhD, director of the NIMH Office of AIDS Programs, said, "this is the first time that a trial of an AIDS therapeutic will be using improvement in mental functioning as the key outcome. We are working toward making progress in treating HIV infection and are looking forward to the collaboration with USC investigators and the results of this study."

Peptide T was developed by NIMH scientists working at the institute's research facilities on the National Institutes of Health campus in Bethesda, Md. The peptide is an artificially produced protein that blocks the attachment of the AIDS virus to human cells by occupying receptors on the cell surface. Its structure is similar to part of the protein coat of HIV.

In the Phase II study, a minimum of 150 men and women who have been infected with HIV will be enrolled in the double-blind, placebo-controlled trial. (Phase II studies are designed primarily to test the efficacy of new therapies.)

The investigators are making special efforts to enroll women and minorities, who have been underrepresented in some earlier AIDS studies.

Initially, participants will receive either Peptide T or placebo for 6 months, and then during the second 6-month period, all study participants will receive the peptide. During the 1-year study, participants may take any medications approved by the Food and Drug Administration for preventing AIDS-related opportunistic infections, and they may continue treatment with antiviral therapies if they are taking them when they enter the study.

Individuals interested in participating in the study should call Charles Hovis, LAC-USC Medical Center, 213/226-4643, or the PHS-sponsored AIDS Clinical Trials Information Service, 1-800-TRIALS-A.

Breast Cancer Screening among Relatives of Women with Breast Cancer

Karen M. Kaplan, MD, Gene B. Weinberg, DrPH, Alden Small, PhD, and Joy L. Herndon, MS

ABSTRACT

Background. National surveys indicate that only 15% to 30% of all women in the general population 50 years of age or older have an annual mammogram.

Methods. We studied first-degree female relatives of women with breast cancer, who are at elevated risk of disease, to describe the breast cancer screening practices of these relatives and to improve their practices through a program of intensive education. We report here the screening behaviors of 2471 relatives of women with breast cancer.

Results. Self-reported behaviors were as follows: 49% performed monthly breast self-examination and 70% had annual breast examinations by a medical professional. Of 983 women 50 years of age or older, 49% had had a mammogram, but only 14% have a mammogram annually. Of women 50 years of age or older who had never had a mammogram, 92% reported never having had one recommended by a medical professional.

Conclusions. Our findings indicate that screening behaviors in relatives of breast cancer patients are not substantially different from those of women in the general population. Enhanced efforts both to educate medical professionals and to encourage women to demand screening mammography are necessary to reduce breast cancer mortality. (*Am J Public Health.* 1991;81:1174–1179)

Introduction

Nearly one in nine women in the United States will have breast cancer in her lifetime. An estimated 175 000 new cases will be diagnosed and an estimated 44 500 deaths from this disease will be recorded during 1991. Breast cancer ranks second only to lung cancer as a cause of cancer mortality in women; yet despite biomedical advances in breast cancer therapy, mortality has remained fairly constant and has even increased slightly in recent years.[1]

Routine breast examination by a physician and breast self-examination (BSE) both have been shown to be more effective than accidental discovery in detecting the disease at an early stage.[2,3] Moreover, detection of early lesions using a combination of professional breast examination and mammography for women ≥50 years of age, and more recently for women 40 to 49 years of age, has been shown to improve survival from breast cancer.[4]

Despite the potential reduction in morbidity and mortality using available breast cancer screening methods, screening measures are underutilized. Recent population-based surveys indicate that 50% of women ≥50 years of age perform monthly BSE, 50% have annual professional breast examinations, and no more than 15% to 30% have annual mammograms.[5–8]

A total of 8% of women 40 years of age or older in the United States have at least one first-degree female relative (mother, daughter, or sister) who has had breast cancer.[9] Nevertheless, little is known about the screening behaviors of these relatives,[10–12] women whose risk of disease is estimated to be twofold to fourfold higher than that for women in the general population.[13] Krischer and colleagues studied the breast cancer screening behaviors of 138 first-degree and 70 second-degree relatives of women with breast cancer and found low prevalence rates of breast cancer screening overall, rates comparable to those of women in the general population.[10] To further investigate this group of high-risk women, the Pennsylvania Cancer Program of the Pennsylvania Department of Health supported six pilot projects from 1984 to 1987. The projects were designed to identify relatives of women with breast cancer, to describe the breast cancer screening behaviors of these relatives, and to improve their behaviors through a program of intensive education.

We report here the screening behaviors of these relatives in five of the pilot projects. We hypothesized that health care providers would more effectively screen women who were known to be at elevated risk of breast cancer and that the prevalence of screening behaviors among this population would be higher than that among women in the general population.

Karen M. Kaplan and Joy L. Herndon were with the Epidemiology Program Office, Centers for Disease Control, Atlanta, Ga. Gene B. Weinberg and Alden Small are with the Pennsylvania Department of Health, Harrisburg, Pa. Dr. Kaplan is now with the Department of Pediatrics, The Pennsylvania State University College of Medicine, Hershey, Pa.

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This paper was submitted to the journal June 20, 1990, and accepted with revisions April 2, 1991.

TABLE 1—Prevalence of Breast Self-Examination (BSE), Professional Breast Examination (BE), and Mammography (MAM) by Selected Characteristics, Relative Breast Cancer Screening Project, Pennsylvania, 1984–1987

Characteristic[a]	Total	Ever BSE (%)	Monthly BSE (%)	Ever BE (%)	Annual BE (%)	Ever MAM (%)	Periodic MAM (%)[b]
Overall	2471	83	49	96	70	37	15[c]
Age (years)							
<40	1059	84	49	96	77	42[d]	NA[e]
40–49	424	86	54	97	70	53	17
>50	983	82	47	96	61	49	14
Race							
White	2362	82	47	96	61	48	15
Non-White	106	79	41	97	74	56	11
Residence							
Urban	1091	84	55	97	76	42	19
Mixed	1268	83	46	95	65	33	13
Rural	112	89	24	96	61	31	4
Relation							
Daughter	1231	85	50	96	74	26	17
Sister	1159	82	49	96	65	47	15
Mother	81	78	38	98	68	54	11
Education (years)							
<12	441	79	44	93	53	39	10
12	1119	84	48	96	69	50	15
>12	858	81	52	98	78	59	23
Income($)							
<20,000	843	79	46	95	61	41	11
20,000–40,000	1001	86	51	96	74	55	17
>40,000	414	85	53	100	80	73	27

[a] Data are not available for all behaviors stratified by all characteristics.
[b] Biennial mammograms in women 40–49 and annual mammograms in women ≥50.
[c] Women ≥40 only.
[d] Women 35–39 only.
[e] NA = not applicable.

Methods

All women diagnosed with breast cancer from 1980 to 1986 were identified from cancer registries in eight Pennsylvania hospitals and from the Pennsylvania Cancer Registry in the south-central region of the state. Only women known to be alive at the time of the study were included. The physician who completed the cancer registry data form was contacted for permission to contact the patient; women whose physicians granted permission received letters describing the study and inviting their participation. Women with breast cancer were asked to identify and give permission to contact their mothers, sisters, and daughters, ≥18 years of age, for enrollment in the study.

Data were collected either by telephone or in face-to-face interviews using a structured questionnaire that included demographic characteristics and information about three breast cancer screening behaviors: performance of BSE, breast examinations by a medical professional, and mammography. All information was self-reported; no attempt was made to verify the reported frequencies of screening behaviors by review of medical records.

Because the majority of participating relatives lived in the area of the hospital and because no specific information on residence was collected, the catchment area of the hospital was used as a surrogate for characterizing residence.

The Statistical Analysis System (SAS Institute, Cary, NC) was used for data analysis. Categorical data were compared using χ^2 and the χ^2 test for trend. In reporting stratified data on screening behaviors, only participants with complete risk factor and outcome information were included. To compute the odds ratio for each of the three screening behaviors while simultaneously controlling for the potentially confounding effects of other characteristics, we used unconditional logistic regression (SAS CATMOD with the CLOGITS option).

Results

Demographics

A total of 4012 women with histologically confirmed breast cancer were identified. At the time of the study, 3229 were still alive and were well enough to participate. The physicians of 2975 (92%) of these gave consent for their patients to be contacted. A total of 2050 women (64%) agreed to participate, and 1862 (58%) were interviewed.

Women with breast cancer identified 3860 relatives, of whom 2865 (74%) were invited to participate. A total of 2540 relatives (66%) agreed to participate, and 2539 were interviewed. Data on 68 of these women (2% of the total group of relatives) who reported that they also had had breast cancer were excluded from the analysis. The analysis thus consists of 2471 women, 64% of the relatives of participating women with breast cancer.

Characteristics of participating relatives are summarized in Table 1. The majority were <50 years of age, White, had completed at least 12 years of education, and had a household income >$20,000. A total of 44% of participants were from urban areas, 51% were from areas of mixed type (urban and rural), and 5% were from rural areas. Information about relatives who declined to participate was not available.

Screening Behaviors

A total of 83% of women have done BSE, and 96% have had at least one breast examination by a medical professional (Table 1). A total of 42% of women 35 to 39 years of age, 53% of women 40 to 49 years of age, and 49% of women ≥50 years of age have had at least one mammogram. A total of 28% of women ≥50 years of age reported having had a mammogram within the previous 12 months.

In order to more accurately distinguish routine screening behaviors from breast examinations or mammography done because of breast abnormalities, we also examined periodic performance of breast cancer screening. Table 1 shows the proportions of women reporting monthly BSE, annual breast examinations from a professional, periodic mammography (biennial mammograms for women 40 to 49 years of age and annual mammograms for women ≥50 years of age) for women overall and stratified by age, race, residence, relationship, education, and income. Younger women, women from urban areas, daughters of women with breast cancer, more highly educated women, and women of higher income more often performed monthly BSE, had annual breast examinations from a professional, and had periodic mammography. Although the numbers of non-White

TABLE 2—Mammography and Breast Examination by a Professional in Women ≥50, Relative Breast Cancer Screening Project, Pennsylvania, 1984–1987

	Mammogram within 12 months		
	Yes	No	Total
Professional breast exam within 12 mo.	No. (%)	No. (%)	No. (%)
Yes	263 (41)	375 (59)	638 (67)
No	6 (2)	315 (98)	321 (33)
Total	269	690	959 (100)

Note: N = 959

TABLE 3—Medical Specialist Doing Breast Examination and Proportion of Their Patients ≥50 Years Having Mammograms, Relative Breast Cancer Screening Project, Pennsylvania, 1984–1987

	Any Mammogram			Annual Mammogram		
Medical specialist	Yes	Total patients	Percentage	Yes	Total patients	Percentage
OB/gyn	194	323	60	66	321	21
Internist	38	67	57	16	67	24
General practitioner	138	356	39	27	356	8
Others[a]	99	178	56	30	178	17

[a] Includes physicians of unknown specialty, surgeons, and oncologists.

women in the study population were small, White women performed BSE and had periodic mammography more frequently. Non-White women more frequently had annual professional breast examinations.

Medical Care Encounters

Of 700 women >40 years of age who had never had a mammogram, 89% reported that a mammogram had never been recommended to them by a medical professional. Stratified by age, 83% of women ages 40 to 49 and 92% of women ≥50 years of age reported never having had a mammogram recommended.

The 75 women who had never had a mammogram, but to whom a mammogram had been recommended, were asked why they had not had one. A total of 19% responded "no particular reason," 17% cited high cost as the primary reason, and 13% said that they planned to have a mammogram.

Of 959 women ≥50 years of age for whom information was available, 67% had had a professional breast examination within the previous 12 months. Of these women, 41% had had a mammogram within that period of time compared with 2% of women who had not had a breast examination within 12 months ($\chi^2 = 163.58$; $P < .001$) (Table 2). Nevertheless, 59% of women who had had a breast examination by a professional within the previous 12 months had not had a mammogram within that period of time.

In the same group of 959 women ≥50, we noted the type of medical specialist usually seen for a breast examination stratified by mammography use and annual mammography use (Table 3). A total of 60% of women who usually have a breast examination by an obstetrician/gynecologist had had a mammogram, compared with 57% seen by internists and 39% who usually have breast examinations by a general practitioner (χ^2 for trend = 34.31; $P < .001$). Moreover, only 8% of women usually seen by a general practitioner for breast examinations have annual mammograms, compared with 21% seen by obstetrician/gynecologists and 24% seen by internists ($\chi^2 = 28.24$; $P < .001$). Some women were seen for breast examinations by surgeons, oncologists, or physicians whose specialties were unknown or unreported. These medical practitioners make up the "other" category. The extent to which the practices of physicians in this category are comparable is not known. The differences by medical specialty persisted when the data were stratified by age groups, education groups, and income groups. When we stratified the data by residence, we found the same differences in mammography use based on the type of physician seen for a breast examination among residents of both urban and mixed (urban and rural) settings. There were too few observations for women living in rural areas to make meaningful comparisons among the three types of residences, however.

Multivariate Analyses

We examined BSE, breast examination by a professional, and annual mammography using unconditional logistic regression. The importance of age, income, education, urban setting, family relation, and type of medical professional seen for a breast examination were all analyzed in connection with the three outcome behaviors mentioned above (Table 4). Controlling for the other variables in the model, monthly BSE was significantly associated with urban residence. For the outcome of annual breast examination by a professional, the variables of most importance when controlling for other factors were younger age, higher income, urban residence, and type of medical professional seen for a breast examination. (Women usually seen by obstetrician/gynecologists were more likely to have annual examinations than women seen by general practitioners.) When these same independent variables were analyzed with annual mammograms as the outcome variable, older age, higher income, urban residence, and type of medical professional seen for a breast examination were the variables of most importance. Women usually seen by obstetrician/gynecologists, internists, or physicians in the "other" category were more likely to have annual mammograms than women usually seen for breast examinations by general practitioners.

Adherence to American Cancer Society Screening Guidelines

We examined the behaviors of women in the two age groups 40 to 49 and ≥50 to determine what proportions adhere to American Cancer Society (ACS) guidelines: monthly BSE; annual breast examinations by a professional; and mammography, biennially for women 40 to 49 and annually for women ≥50 years of age.[14] Seventeen percent of women ages 40 through 49 have biennial mammograms, and 14% of women ≥50 have annual mammograms (Table 1). A total of

TABLE 4—Prevalence of Annual Mammogram (MAM), Annual Professional Breast Examination (BE), and Monthly Breast Self Examination (BSE) by Selected Characteristics, Logistic Regression Model, Relative Breast Cancer Screening Project, Pennsylvania, 1984–1987

Variable	Annual MAM		Annual BE		Monthly BSE	
	OR	95% CI	OR	95% CI	OR	95% CI
Relation (base = daughters)						
Sisters	1.743	1.110–2.735	1.276	0.943–1.725	0.906	0.703–1.168
Mothers	1.130	0.452–2.826	1.519	0.798–2.891	0.516	0.289–0.921
Age (years) (base = <40)						
40–49	3.975	2.352–6.718	0.706	0.507–0.982	1.446	1.096–1.910
50 and older	5.212	3.011–9.021	0.494	0.353–0.691	1.156	0.866–1.543
Residence (base = urban)						
Mixed	0.661	0.475–0.920	0.697	0.556–0.875	0.695	0.575–0.842
Rural	0.141	0.018–1.087	0.693	0.417–1.151	0.320	0.193–0.531
Education (base = <12						
12	0.940	0.593–1.488	1.202	0.902–1.600	0.907	0.686–1.200
>12	1.200	0.709–2.030	1.400	0.985–1.991	0.989	0.719–1.361
Income (base = <$20,000)						
$20,000–$40,000	1.794	1.214–2.653	1.394	1.098–1.770	1.202	0.973–1.486
Over $40,000	2.711	1.679–4.375	1.799	1.277–2.533	1.147	0.869–1.512
Physician specialty (base = general practice)						
Ob/gyn	1.774	1.145–2.748	1.608	1.240–2.086	1.165	0.923–1.472
Internal medicine	2.784	1.417–5.471	1.783	0.977–3.253	0.856	0.514–1.426
Other	2.751	1.666–4.541	1.009	0.727–1.400	1.113	0.820–1.510
Total n	2003		2024		2068	

Note: OR = odds ratio; CI = confidence interval.

TABLE 5—Prevalence of Breast Self-Examination (BSE), Professional Breast Examination (BE), and Mammography (MAM), Pennsylvania, 1984–1987, vs Studies of Women in the General Population

Behavior	Percentage Pennsylvania	Percentage General Population
Monthly BSE	49	47–50[8,9]
Annual BE	70	50–55[5–7]
MAM[a]	50	40–67[7–9,16]
Annual MAM[b]	14	15–30[6–8]
MAM ≤1 yr[b]	28	20–41[7,15,17,21]

[a] Women ≥40 years of age.
[b] Women ≥50 years of age.

41% of women 40 to 49 and 32% of women ≥50 adhere to the recommended screening for two of the three practices, monthly BSE, and annual professional breast examinations. Only 11% and 8%, respectively, adhere to all three screening guidelines for their age group.

We compared our findings with those of other surveys of breast cancer screening among women in the general population (Table 5). The prevalence of monthly BSE and mammography use reported in our study is similar to that of women in the general population.[5–8,15–19] The prevalence of annual professional breast examination, 70%, is higher than generally reported.[5–7,9] The proportion of women in the general population who practice a combination of these three screening methods is not known.

Discussion

Our findings indicate that BSE and mammography use in relatives of women with breast cancer, who are at elevated disease risk, do not differ substantially from the behaviors of women in the general population. Despite the higher rates of professional breast examinations reported for this population, barriers to more widespread use of screening mammography exist. Our data do not support the hypothesis that physicians screen selectively and comprehensively those women known to be at elevated risk of breast cancer. Nearly 90% of women who had never had a mammogram reported that a medical professional had never recommended one.

Older women, women of lower socioeconomic status, and women living in rural areas were less likely to report adherence to current ACS breast cancer screening guidelines. Similar risk factors for inadequate preventive health practices in general and for breast cancer screening in particular have been found in national surveys.[9,19–21]

Not surprisingly, we found that women who reported having had a recent professional breast examination more often reported having had a recent mammogram. Yet the majority of women ≥50 seen for a breast examination within the previous year who would have been eligible for screening mammography were not screened, most often because their physicians had not recommended it.

We found differences in compliance with guidelines for mammography use among medical specialties: women who usually had breast examinations by internists or obstetrician/gynecologists were more likely to have mammograms than women who usually had breast examinations by general practitioners. Reasons for the apparent differences in practices among medical specialists are not clear, but such differences may in part reflect type or recency of medical training or participation in continuing medical education. It is also not known whether women in the study population who have breast examinations by general practitioners were less likely to have health insurance that covers screening mammography than were women seen by internists or obstetrician/gynecologists. Bergner and colleagues[22] found that recent medical school graduates and medical school faculty members were more likely to perform a series of cancer screening procedures among asymptomatic patients over the age of 50 for whom they provided primary care. In their survey, obstetrician/gynecologists followed by oncologists most often performed breast examinations and screening mammograms. Information on years since graduation from medical school or faculty status was not available for the physicians of participants in our study.

The failure of physicians to follow established guidelines for breast cancer screening in asymptomatic women was documented in a 1989 Ohio survey of more than 1000 practicing primary care physicians.[23] The survey found that although 98% advise their patients to do BSE, and 78% followed published ACS guidelines for periodic breast physical examinations, only 37% followed guidelines for screening mammograms in asymptomatic women.[23] A similar survey conducted in 1984 found 80% and 11% of physicians adhering to ACS guidelines for breast physical examinations and screening mammography, respectively.[24]

The National Cancer Institute (NCI) has developed the following objective for breast cancer screening for the year 2000: 80% of women 50 to 70 years of age will have both annual breast examinations by a medical professional and annual mammograms.[25] The authors of *Healthy People 2000* suggest that the NCI breast cancer screening goals be incorporated into the Nation's Year 2000 Health Objectives.[26] To achieve these goals, enhanced educational efforts are needed, particularly in promoting mammography. These efforts should target both primary health care providers and women ≥50 years of age. Medical school, residency training, and continuing medical education programs should include instruction on the current approaches to breast cancer screening. The 1989 survey of practicing primary care physicians suggested that misinformation about radiation risks from mammography may deter some physicians from ordering screening mammograms for their patients.[23] In addition, women should be encouraged to demand screening mammography, an approach that has shown utility in stimulating physicians to provide other preventive services.[27–29]

Several limitations of the study design should be noted. First, participation rates were low for women with breast cancer (58%) and their relatives (64%); yet there was no information by which to compare characteristics of participants and nonparticipants. We believe that nonparticipants are unlikely to have better screening behaviors than participants.

Second, in order for women with breast cancer to provide access to their relatives, only those women still alive at the time of the study were eligible breast cancer patients. Whether relatives of women who have died from breast cancer differ from other relatives in their screening behaviors is not known. Further research is needed to better understand factors such as stage of disease at diagnosis, extent of disease, or type of therapy, all of which may affect preventive behaviors in asymptomatic relatives.

Third, all information in the study was self-reported and thus subject to recall bias on the part of the respondents. No attempt was made to verify the reported frequency of professional breast examinations or mammography use by review of medical records. It is therefore possible that some groups of women are more likely to recall breast cancer screening practices better than others.

Fourth, the population studied was primarily White and of relatively high socioeconomic status; the findings may be neither comparable to findings of other population surveys of breast cancer screening behaviors nor generalizable to all populations. Additional studies should focus on the breast cancer screening practices of non-White women and women of lower socioeconomic status who are at elevated risk of breast cancer.

Despite these limitations, our data confirm the findings of previous surveys of women's breast cancer screening behaviors: screening remains underutilized.[5–9,15–18] Whether any of these surveys, including our own, actually measure women's behaviors as opposed to those of their health care providers is not clear. In either case, intensified efforts are needed to enhance the early detection of breast cancer in order to reduce mortality from this disease. □

Acknowledgments

Russell A. Localio, MPH, MS, provided statistical support and consultation. Drs. David T. Dennis, Robert A. Gunn, Robert F. Anda, and Leslie Boss provided helpful comments on earlier versions of the manuscript. This study was presented, in part, at the 117th annual meeting of the American Public Health Association, Chicago, Ill, October 22–26, 1989.

References

1. Boring CC, Squires TS, Tong T. *Cancer Statistics 1991.* CA 1991;41:19–36.
2. Greenwald P, Nasca PC, Lawrence CE, et al. Estimated effect of breast self-examination and routine physician examinations on breast cancer mortality. *N Engl J Med.* 1978;299:271–273.
3. Foster RS, Lang SP, Costanza MC, Worden JK, Haines CR, Yates JW. Breast self-examination practices and breast-cancer stage. *N Engl J Med.* 1978;299:265–270.
4. Chu KC, Smart CR, Tarone RE. Analysis of breast cancer mortality and stage distribution by age for the health insurance plan clinical trial. *JNCI.* 1988;80:1125–1131.
5. Opinion Research Corporation. *Breast Cancer: A Measure of Progress in Public Understanding.* Princeton, NJ; 1984.
6. American Cancer Society: 1983 *Survey of Public Awareness and Use of Cancer Detection Tests.* Princeton, NJ: Gallup Organization; 1983.
7. American Cancer Society: 1987 *Survey of Public Awareness and Use of Cancer Detection Tests.* Princeton, NJ: Gallup Organization; 1988.
8. Centers for Disease Control. Use of mammography—United States, 1990. *MMWR.* 1990;39:621–630.
9. Dawson DA, Thompson GB. Breast cancer risk factors and screening: United States, 1987. *Vital Health Stat.* 1989;10:13–26.
10. Krischer JP, Cook B, Weiner RS. Identification and screening of women at high risk of breast cancer. *Cancer Detect Prev.* 1988;13:65–74.
11. Taplin S, Anderman C, Grothaus L. Breast cancer risk and participation in mammographic screening. *Am J Public Health.* 1989;79:1494–1498.
12. Dworsky R, Paganini-Hill A, Spicer D: Breast cancer screening and education in high risk women. *Prog Clin Biol Res.* 1989;293:115–124.
13. Petrakis NL, Ernster VL, King M-C: Breast. In: Schottenfeld D, Fraumeni JF, eds. *Cancer Epidemiology and Prevention.* Philadelphia, Pa: WB Saunders; 1982:855–870.
14. Dodd GR, Fink DJ, Murphy GP: Breast cancer detection and community practice: executive summary report of a workshop cosponsored by the General Motors Cancer Research Foundation and the American Cancer Society. Ca, 1989;39:227–229.
15. Hayward RA, Shapiro MF, Freeman HE, Corey CR. Who gets screened for cervical and breast cancer? Results from a new national survey. *Arch Intern Med.* 1988;148:1177–1181.
16. Centers for Disease Control. Trends in screening mammograms for women 50

years of age and older—behavioral risk factor surveillance system, 1987. *MMWR.* 1989;38:137–140.

17. Centers for Disease Control. Provisional estimates from the national health interview survey supplement on cancer control—United States, January–March 1987. *MMWR.* 1988;37:417–420, 425.
18. NCI Breast Cancer Screening Consortium. Screening mammography: a missed clinical opportunity? *JAMA.* 1990;264:54–58.
19. Reeder S, Berkanovic E, Marcus AC. Breast cancer detection behavior among urban women. *Public Health Rep.* 1980;95:276–281.
20. Woolhandler S, Himmelstein DU. Reverse targeting of preventive care due to lack of health insurance. *JAMA.* 1988;259:2872–2874.
21. Dawson DA, Hendershot GE, Bloom B. Trends in routine screening examinations. *Am J Public Health* 1987;77:1004–1005.
22. Bergner M, Allison CJ, Diehr P, Ford LG, Feigl P. Early detection and control of cancer in clinical practice. *Arch Intern Med.* 1990;150:431–436.
23. Holleb AI. 1989 survey of physicians' attitudes and practices in early cancer detection. *CA.* 1990;40:77–101.
24. Holleb AI. Survey of physicians' attitudes and practices in early cancer detection. *CA.* 1985;35:197–213.
25. Greenwald P, Sondik EJ, eds. Cancer control objectives for the nation 1985–2000. *NCI Monogr.* 1986;2:27–32.
26. Stoto MA, Behrens R, Rosemont C, eds. *Healthy People 2000: Charting the Course.* Washington, DC: National Academy Press; 1990:186–190.
27. Korsch BM, Gozzi EK, Francis V. Gaps in doctor-patient communication I: doctor-patient interaction and patient satisfaction. *Pediatrics.* 1968;42:855–871.
28. Roter DL. Patient participation in the patient-provider interaction: the effects of patient question-asking on the quality of interaction, satisfaction, and compliance. *Health Educ Monogr.* 1977;5:281–315.
29. Schulman BA. Active patient orientation and outcomes in hypertensive treatment: application of a socio-organizational perspective. *Med Care.* 1979;17:267–280.

New Insights into Sudden Infant Death Syndrome

Results from a study published in the May 1 *American Journal of Epidemiology* suggest that certain labor and delivery events may increase the risk of sudden infant death syndrome (SIDS).

SIDS is the leading cause of mortality in the United States for infants 1 to 12 months old, accounting for one to three deaths per 1000 live births. SIDS has been called "a diagnosis of exclusion" because its victims can only be identified through an autopsy that rules out all other explainable causes of death.

This study "is one of the first analytic studies to focus on the intrapartum period and risk factors for SIDS," says lead author Germaine M. Buck, PhD, clinical assistant professor, Department of Social and Preventive Medicine, School of Medicine and Biomedical Sciences, Buffalo, NY.

Buck and her team found a sevenfold increased risk for SIDS associated with vaginal breech delivery and more than twice the risk for SIDS when mothers were in labor 16 hours or longer. Their study population was selected from a 1974 cohort of live births from Upstate New York that included 132 948 resident mothers. Data were abstracted from hospital delivery and vital records for 148 autopsied cases and 355 matched controls.

"The majority of breech SIDS infants were single footling deliveries (a rarer type of breech presentation with the baby emerging one foot first). The more common form of breech delivery called 'frank' presentation, with the baby emerging buttocks first, was not associated with an increased risk for SIDS," Buck says.

"We suspect that the single footling breech presentation may be an indicator for an earlier problem in the fetus' development, while a breech delivery, per se, is not the direct cause of the syndrome," she says. This speculation concurs with previous studies that have suggested SIDS victims may be compromised before birth. As Buck explains, "SIDS babies may have experienced a prior central nervous system insult in utero since they often show signs of subtle growth retardation, including reduced birth weight and length." These effects could be explained by altered blood flow or oxygen supply to the developing fetus, Buck says, but not by labor or delivery events.

"The association between long labor and SIDS must be viewed conservatively," Buck adds. "There is a lot of subjectivity in the woman's reporting of how long she has been in labor when she is first seen in the hospital."

According to Buck, the incidence of SIDS has not diminished as prenatal care has improved in the last several decades. "SIDS is a chief contributor to this nation's high infant mortality rate, and further effort to understand this syndrome should be a public health mandate," she says.

Factors Influencing the Formation of New Human Coronary Lesions: Age, Blood Pressure, and Blood Cholesterol

Wendy J. Mack, PhD, and David H. Blankenhorn, MD

ABSTRACT

Background. The Cholesterol Lowering Atherosclerosis Study, a controlled angiographic trial, has reported that new native coronary artery lesions are significantly reduced by aggressive blood lipid lowering therapy with colestipol plus niacin. To study factors relevant to primary atherosclerosis prevention, we have conducted an epidemiologic analysis of new native coronary lesion formation in placebo-treated patients.

Methods. Univariate and multivariate logistic regression procedures were used to examine age at entry into the study, number of years since bypass, body weight, diastolic and systolic blood pressure, plasma total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, non-HDL-cholesterol, and apolipoproteins A-I,B, and C-III.

Results. Significant univariate protective factors were older age ($P<.006$), reduction of total plasma cholesterol ($P<.040$), and systolic ($P<.024$) and diastolic ($P<.022$) blood pressure. Significant multivariate protective factors were older age ($P<.005$) and reduction in systolic blood pressure ($P<.021$). Blood pressure effects were not associated with use of specific antihypertensive agents.

Conclusions. These data provide additional support for the control of hypertension and reduction of blood cholesterol level for primary and secondary ischemic heart disease prevention. They also indicate the existence of a population at high risk for early coronary lesion formation and the need for improved means to identify such individuals prior to the onset of clinical manifestations of ischemic heart disease. (*Am J Public Health.* 1991;81:1180–1184.)

Introduction

CLAS (Cholesterol Lowering Atherosclerosis Study) was a randomized, placebo-controlled angiographic trial testing the hypothesis that blood lipid lowering with colestipol plus niacin would slow or reverse the atherosclerotic process. CLAS was a secondary prevention trial involving men, ages 40 to 59 years, who had had previous aortocoronary bypass surgery. CLAS angiograms were evaluated for vessel closure, changes in degree of stenosis, and formation of new lesions. We have previously reported significant treatment benefits in overall coronary status of both native vessels and bypass grafts, including reduction of formation of new lesions.[1] Following publication of CLAS results, angiographic trials testing nifedipine[2] and dipyridamole plus aspirin[3] have also reported reduced formation of new coronary lesions, which suggests that pathways early in atherogenesis may be beneficially modified by several different types of therapy. Studies of atherogenesis in animal models and cultured cell systems suggest a variety of ways in which factors initiating lesion formation might differ from those that cause progression of established lesions. The relationship of risk factors to prevalence of human coronary lesions[4] and change in established coronary lesions[5–20] has been extensively studied by angiography, but there are no risk analyses for new coronary lesion formation. To explore risk factor effects on human new coronary lesion formation we conducted an analysis of risk for disease with nondisease in placebo-treated CLAS subjects by comparing subjects who developed new coronary lesions with those who did not. The influence of diet on new lesion formation has been reported previously.[21] We now address the association of clinical variables with the development of new lesions.

Methods

Study Population

Subjects were nonsmoking males, ages 40 to 59 years, with progressive symptomatic atherosclerosis who had had previous coronary bypass surgery. Screening procedures, criteria for exclusion, standard visit protocols, and basic intervention goals were reported earlier.[22] One hundred eighty-eight subjects were randomized into two treatment groups of 94 each; 162 completed the study, 82 of whom were placebo-treated. Average age at entry of all placebo subjects was 54.5 ± 0.5 (SEM) and average blood pressure was 123 mm Hg ± 1.5 (SEM)/80 ± 1.0 (SEM). Thirty percent never smoked and 70% had quit smoking more than 6 months before trial entry. Twenty-four-hour urine specimens were examined for nicotine and cotinine every 6 months to monitor nonsmoking status. Dietary goals for the placebo group were as follows: total fat calories to provide 26% of energy, less than 5% of energy from saturated fatty acids, 10% of energy from polyunsaturated fatty acids, and less than 250 mg cholesterol per day. One-on-one diet counseling was conducted at each clinic

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This paper was submitted to the journal July 30, 1990, and accepted with revisions March 6, 1991.

visit with a review of fat and cholesterol intake in computerized 7-day food records from the preceding week. Levels of physical activity were moderate as judged from questionnaires and estimates of energy expenditure from records of caloric intake for the 7 days preceding each clinic visit.

Data Collection

Angiographic. Baseline coronary angiography was performed immediately before randomization. A repeat angiogram was performed at 2 years by the same angiographer, who was blinded to the treatment assignment. Coronary angiogram evaluation procedures have been described in detail previously.[1,22] All lesions greater than 20% diameter stenosis were identified. Film pairs showing identical coronary artery views were mounted side by side on two Vanguard projectors and viewed simultaneously by two expert angiographers who did not know the subject's demographic and clinical characteristics, treatment assignment, or temporal order of the angiograms. The angiographers were not shown ventriculograms and other angiograms. Working independently, the two angiographers identified all visible lesions in one film (assigned at random), recorded this information, and then shared it to reach a consensus. In this manner a total of 1831 lesions were recognized and evaluated, 82% of which were identified independently by both panelists. For the 1501 lesions identified independently by both panelists, agreement within ±10% stenosis occurred in 76%. After reaching a consensus on the first film, panelists examined the second film alongside the first and compared all lesions first separately and then together. After all film pairs were evaluated, the code for film order was broken and lesions recorded in a later angiogram, but not in a first, were tabulated.

Body Weight, Blood Pressure, Plasma Lipids, and Medication History. Body weight, blood pressure, and plasma lipids were measured at every visit.[22] Blood lipids and lipoprotein cholesterol levels were determined by Lipid Research Clinics methodology standardized against reference materials supplied by the Centers for Disease Control.[23] LDL-cholesterol levels were calculated with the equation of Friedewald et al.[24] Baseline levels were the means of the first three screening visits. Postrandomization visits were scheduled every month for the first 6 months and bimonthly thereafter. On-trial values were the average of all postrandomization visits weighted by the scheduled interval between treatment visits. Apolipoprotein determinations were performed by the Oklahoma Foundation for Medical Research.[22] All medications, including over-the-counter drugs, were recorded at semiannual visits (0, 6, 12, 18, and 24 months after randomization).

At study entry 36% of the 82 CLAS placebo subjects reported use of antihypertensive medications and 93% of this group remained on these agents during the trial. The most common drugs and dosage were propranolol 40 mg/day (33% of subjects), hydrochlorothiazide 50 mg/day (13%), and dyazide 50 mg/day (10%). Eight percent of CLAS placebo subjects reported one or more on-trial uses of either nifedipine, diltiazem, or verapamil. Eighty percent of subjects took either aspirin, Ascriptin, or acetaminophen on one or more occasions during the trial.

Statistical Analyses. Univariate logistic regression procedures were used to determine which independent variables were predictive of the development of new lesions. Potentially predictive independent variables included age at entry into the study, number of years since bypass, body weight, diastolic and systolic blood pressure, plasma total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, non-HDL-cholesterol, and apolipoproteins A-I, B, and C-III. Age and years since bypass were analyzed by values at baseline. All other variables were analyzed by values at baseline, as well as mean on-trial, to explore the temporal relationship of these variables to lesion formation. In addition, the difference (baseline minus mean on-trial) was analyzed to assess the relationship between change in these variables and new lesion formation. We chose to compare baseline to mean on-trial values of each independent variable for two reasons. First, given the relatively small sample size of this study and the fact that each subject contributed a potential of 15 on-trial data points for each variable, it was not feasible to assess the significance of each variable at each time point. Second, given the short time period of this study (2 years) relative to the several decades of life, over which atherosclerotic disease develops, we felt that assessing the association of overall on-trial changes relative to prerandomization measures provided a more meaningful analysis. For each independent variable, univariate odds ratios (OR) and associated 95% confidence intervals (CI) were computed[25]; all *P* values for trends in relative risk are based on two-tailed tests. Stepwise logistic regression was used as a final step to arrive at a multivariate model of statistically independent predictors of new lesion formation.

Results

Of the 82 subjects in the placebo group, 18 had developed new lesions in the 2-year period between angiograms, as previously reported.[1] Thirteen among the 18 subjects with new lesions had a single new lesion, 3 had two new lesions, and 1 each had three and four new lesions. Among the 26 new lesions, 15 were in ungrafted patent arteries, 1 was proximal to complete occlusion in an ungrafted artery, 6 were proximal to open grafts, 2 were distal to open grafts, and 2 were distal to closed grafts. Nine of the new lesions were between 20% and 29% diameter stenosis and 6 were between 30% and 39%. Two lesions were 40% diameter stenosis, 4 were 50%, and 1 each were 60%, 70%, 75%, 80%, and 90% stenosis.

With subjects classified by presence or absence of new lesions, mean values of major clinical variables are presented in Table 1. Descriptively, compared to subjects who did not develop new lesions, subjects who developed new native lesions were usually younger at study entry, had recently undergone bypass surgery, had lost less weight on-trial, and had increased both systolic and diastolic blood pressure while on-trial. Those who developed new lesions also showed minor changes in total cholesterol and LDL-cholesterol (less than 1% decrease in both), a 2% decrease in non-HDL-cholesterol, a 5% increase in HDL-cholesterol, and a 10% decrease in triglycerides. In comparison, CLAS placebo subjects who did not develop new lesions demonstrated larger decreases in total, LDL, and non-HDL-cholesterol (6%, 7%, and 7%, respectively), no change in HDL-cholesterol, and an 8% decrease in triglycerides. Subjects developing new native lesions also had a lower average baseline value of apolipoprotein B (average 111.1 mg/dL compared to 124.1 mg/dL in subjects not developing new lesions) and demonstrated an 8% increase in this variable while on-trial. Subjects who did not develop new lesions showed no average on-trial change in apolipoprotein B. Table 1 also presents the univariate ORs and 95% CIs for each variable. All variables have been divided by their sample standard deviation (SD) so that the resulting ORs represent the relative risk for a 1 SD increase in that variable.

TABLE 1—Independent Variable Means by Lesion Group

Variables	New Lesions Mean ± SD (n = 18)	No New Lesions Mean ± SD (n = 64)	SD[a]	OR[b]	95% CI
Age at study entry	51.7 ± 5.0	55.2 ± 15.7	4.4	0.5	0.3–0.8
Years since bypass	2.4 ± 1.5	2.9 ± 2.3	2.3	0.7	0.4–1.3
Body weight (lbs)					
Baseline	178.3 ± 16.7	177.7 ± 23.3	21.9	1.0	0.6–1.7
On-trial	178.1 ± 16.3	176.8 ± 25.2	23.4	1.1	0.6–1.8
Difference	0.2 ± 6.1	0.9 ± 5.8	5.9	0.9	0.5–1.5
Diastolic BP (mm Hg)					
Baseline	80.8 ± 9.3	80.3 ± 8.3	8.5	1.1	0.6–1.8
On-trial	83.4 ± 7.3	80.1 ± 7.1	7.3	1.6	0.9–2.9
Difference	−2.6 ± 4.5	0.2 ± 4.2	4.4	0.5	0.3–0.9
Systolic BP (mm Hg)					
Baseline	118.8 ± 11.2	124.7 ± 12.0	12.0	0.6	0.3–1.1
On-trial	121.4 ± 10.2	123.0 ± 11.5	11.2	0.9	0.5–1.5
Difference	−2.7 ± 7.1	1.6 ± 6.3	6.9	0.5	0.3–0.9
Plasma total cholesterol (mmol/L)					
Baseline	6.3 ± 1.0	6.3 ± 0.9	0.9	1.0	0.6–1.6
On-trial	6.2 ± 1.2	5.0 ± 0.8	0.9	1.3	0.8–2.1
Difference	0.1 ± 0.5	0.3 ± 0.5	0.5	0.5	0.3–1.0
LDL-cholesterol (mmol/L)					
Baseline	4.3 ± 0.8	4.4 ± 0.8	0.8	0.9	0.5–1.5
On-trial	4.2 ± 1.0	4.1 ± 0.7	0.8	1.2	0.7–2.1
Difference	0.0 ± 0.5	0.3 ± 0.5	0.5	0.6	0.3–1.0
HDL-cholesterol (mmol/L)					
Baseline	1.1 ± 0.2	1.1 ± 0.3	0.2	0.8	0.5–1.3
On-trial	1.1 ± 0.2	1.1 ± 0.2	0.2	1.0	0.6–1.6
Difference	−0.1 ± 0.1	0.0 ± 0.1	0.1	0.7	0.5–1.1
Non-HDL-cholesterol (mmol/L)					
Baseline	5.2 ± 1.0	5.1 ± 0.9	0.9	1.0	0.6–1.7
On-trial	5.1 ± 1.3	4.8 ± 0.8	0.9	1.3	0.8–2.1
Difference	0.1 ± 0.5	0.3 ± 0.5	0.5	0.6	0.3–1.0
Triglycerides (mmol/L)					
Baseline	2.0 ± 1.0	1.7 ± 1.0	1.0	1.3	0.8–2.1
On-trial	1.8 ± 0.9	1.5 ± 0.9	0.9	1.3	0.8–2.1
Difference	0.2 ± 0.3	0.1 ± 0.4	0.4	1.1	0.7–1.9
Apo-A-I (mg/dL)					
Baseline	114.9 ± 15.4	115.6 ± 19.0	18.2	1.0	0.6–1.6
On-trial	123.8 ± 11.6	121.9 ± 15.4	15.7	1.1	0.7–1.9
Difference	−8.9 ± 14.9	−6.3 ± 15.8	15.6	0.9	0.5–1.4
Apo-B (mg/dL)					
Baseline	111.1 ± 25.7	124.1 ± 28.7	26.9	0.6	0.3–1.0
On-trial	119.8 ± 24.2	124.1 ± 22.2	22.6	0.8	0.5–1.4
Difference	−8.8 ± 21.1	0.0 ± 21.0	21.2	0.6	0.4–1.1
Apo-C-III (mg/dL)					
Baseline	5.6 ± 2.8	4.6 ± 2.1	2.3	1.5	0.9–2.5
On-trial	5.7 ± 3.2	4.7 ± 2.5	2.6	1.4	0.8–2.2
Difference	0.0 ± 1.7	−0.1 ± 1.4	1.4	1.1	0.6–1.8

Note. Difference = baseline minus mean ontrial value; CI = confidence interval.
[a] SD = Standard deviation for entire sample (N = 82).
[b] Odds ratio = relative risk per SD change of independent variable (relative to no change).

Independent variables showing univariately significant trends in relative risk for new lesion formation are displayed in more detail in Table 2. To show the trend in relative risk, each variable was categorized into four levels using the sample quartiles, and ORs were estimated for each level relative to the first quartile.

Age at entry into the study was protective for new lesions; thus, older men were less likely to develop new lesions. Dividing the total group of 82 men at the median age (54.5 years), the risk of new lesion development for men above the median was 0.2 times that of men below or at the median age (95% CI = 0.1–0.7). Conversely, the risk for new lesion formation in men younger than 55 years at study entry was 4.8 times that of older men. Inclusion of other significant independent clinical and diet variables[21] did not alter the significance of the influence of age on lesion development.

Changes in both diastolic and systolic blood pressure and total plasma cholesterol from baseline to mean on-trial values were also significant univariate predictors of new lesions (Table 2). Decreases in blood pressure and total plasma cholesterol from baseline to on-trial conferred lower risk for development of new atherosclerotic lesions. Other independent variates, including HDL-, LDL-, and non-HDL-cholesterols, apolipoproteins A-I, B, and C-III, triglycerides, body weight, and years since bypass, were not significantly associated with new lesion formation. Finally, variables indicating whether or not a subject had used platelet-active medications, calcium channel blockers, or other antihypertensive medications were not significantly related to new lesion formation.

To investigate the differential effects of lipids by age, interactions between a dichotomized age variable (dichotomized at the median age at study entry) and plasma lipids measured at baseline and on-trial were included in the logistic regression procedure. Two significant interactions emerged in these analyses. The first interaction was an age-on-trial HDL-cholesterol interaction ($\chi^2 = 3.96$; df = 1; $P = .046$), with the parameter estimate indicating a protective effect for HDL-cholesterol confined to subjects older than 55 years. The interaction between age and on-trial triglycerides was also significant ($\chi^2 = 7.00$; df = 1; $P = .01$) with an increased risk for lesion development due to increased triglyceride level occurring in the older age group. In both cases, the main effect of age remained significant after inclusion of these interactions.

To determine which of the univariately significant variables were independently predictive of new native lesions, a stepwise logistic regression analysis was performed (Table 3). Age at study entry was the first independent variable to enter the model; the risk for development of new native lesions was decreased by a factor of 0.4 for every 4.4 years (ISD) of age. After adjusting for age, the change in systolic blood pressure from baseline to on-trial was also significantly predictive of new lesion formation; each 7 mm Hg decrease in systolic blood pressure was associated with the risk of new lesions being decreased by a factor of 0.5. No other variables were significantly related to lesion development after inclusion of these two variables.

Of the 18 subjects who had developed new lesions, 11 had new lesions in native arteries with related grafts, while 7 had new lesions in native arteries without a related graft. These two subgroups were separately compared to the 64 subjects who did not develop new lesions. Parameter estimates for the final model (age at entry into study and change in systolic blood pressure) did not change from those displayed in Table 3, suggesting that these effects are the same in native arteries with and without related bypass grafts.

Discussion

Our analyses are focused on the appearance of new lesions visible by angiography because these indicate an increase in the extent of raised intrusions into the coronary lumen, the most significant single anatomic predictor of ischemic heart disease (IHD) at autopsy[5] We are concerned primarily with relationships within the placebo group because new lesions were more common here and because these relationships are more relevant to primary IHD prevention than those among drug-treated subjects. Reduction of new lesions in secondary prevention has merit, but reduction of new lesions in primary prevention could benefit many more individuals and might lead to population-wide control of IHD. We have previously reported reduction of new lesions in secondary prevention in CLAS through aggressive treatment with colestipol plus niacin.[1] This treatment reduced average total plasma cholesterol from 6.36 to 4.65 mmol/L and reduced the relative risk of new lesions by a factor of 0.4 ($P = .03$). We have also reported an analysis of diet records of CLAS placebo subjects relevant to primary prevention that indicated that reduction of new lesions might be accomplished by reducing dietary fat.[21] We now report an additional analysis of risk factors relevant to primary prevention that have significant effects on new lesion formation independent of diet. In CLAS placebo subjects, risk for new lesions was predicted by age, on-trial reduction of total plasma cholesterol, and on-trial reduction of both systolic and diastolic blood pressure. When these four univariate predictors competed in stepwise logistic regression, age and change in systolic blood pressure were found to be significant. The effect of age is significant in analyses that pool drug subjects with placebo.

TABLE 2—Univariately Significant Predictors of New Lesion Formation[a]

	OR	95% CI	P trend
Age at entry to study, y			
40 to 52	1.0		.006
>52 to 54.5	0.3	(0.1, 1.1)	
>54.5 to 58	0.1	(0.02,0.6)	
>58	0.1	(0.03,0.8)	
Change in total blood cholesterol (mmol/L)			
≤0	1.0		.040
>0 to 0.2	0.8	(0.2, 2.9)	
>0.2 to 0.6	0.6	(0.2, 2.5)	
>0.6	0.2	(0.1, 1.4)	
Change in diastolic blood pressure (mmHg)			
≤−4	1.0		.022
>−4 to −1	0.4	(0.1, 1.5)	
>−1 to 3	0.3	(0.1, 1.5)	
>3	0.2	(0.04,1.1)	
Change in systolic blood pressure (mmHg)			
≤−3	1.0		.024
>−3 to 1	0.6	(0.2, 2.3)	
>1 to 5	0.4	(0.1, 1.8)	
>5	0.2	(0.04,1.3)	

Note. OR = odds ratio; CI = confidence interval.
[a]Change variables = baseline minus mean on-trial.

TABLE 3—Stepwise Logistic Regression

Variable	OR[a]	95% CI[b]	P
Age at study entry	0.4	0.2–0.8	.005
Change in systolic blood pressure	0.5	0.3–0.9	.021

[a]Odds ratio interpreted as change in risk per standard deviation of age or standard deviation change in systolic blood pressure.
[b]CI = confidence interval.

The multivariate stepwise model associated with on-trial change in systolic blood pressure and age indicates that reduction of systolic blood pressure by 10 mm Hg reduced the risk of the new lesion development by 60% in men of median age 54.5 years. It is important to note that the principal antihypertensive agents used in CLAS placebo subjects were propranolol, hydrochlorothiazide, and dyazide, not slow calcium channel antagonists. When variables indicating whether or not a subject had taken calcium channel blockers or other antihypertensive medications were introduced into logistic regression, they were not significantly related to new lesion formation. We conclude that reduction of blood pressure level per se, rather than the agent used to produce the reduction, was the significant influence in CLAS placebo subjects. Thus, modest changes in blood pressure perceptibly altered atherogenesis at the stage of raised lesion development. This finding is compatible with the large body of epidemiologic evidence for long-term effects of blood pressure on population IHD rates.[27,28] Trials of antihypertensive therapy such as the Multiple Risk Factor Intervention Trial[29] may have failed to show reduced IHD rates because benefits from reduced new lesion formation were masked by catastrophic events in existing coronary lesions.

In summary, placebo group data from CLAS indicate that formation of new intrusive coronary lesions is significantly influenced by age and change in blood cholesterol and blood pressure level. The effects of blood pressure and blood cholesterol are compatible with findings from IHD epidemiology and confirm that it is appropriate to reduce these risk factors for primary IHD prevention. Age was observed to have a negative effect, with more young subjects showing new lesion formation. This indicates the need for improved methods to identify young individuals who form new coronary lesions at accelerated rates. ☐

Acknowledgments

This work was supported by National Heart, Lung, and Blood Institute Program Project Grant HL23619 and The Upjohn Company.

The authors wish to acknowledge Jo Darnall for assistance in the preparation of the manuscript, the CLAS Clinical, Biostatistical, and Lipid Laboratory Staff, and the Coronary Film Panelists: Drs. Miguel E. Sanmarco, George G. Rowe, Peter R. Mahrer, Ivan L. Bunnell, William J. French, C. Richard Conti, J. Michael Criley, Harold T. Dodge, David G. Greene, W. David Johnston, K. Ramaswamy, Douglas K. Stewart, and Bonnie H. Weiner.

References

1. Blankenhorn DH, Nessim SA, Johnson RL, Sanmarco ME, Azen SP, Cashin-Hemphill L. Beneficial effects of combined colestipol-niacin therapy on coronary atherosclerosis and coronary venous bypass grafts. *JAMA*. 1987;257:3233–3240.
2. Lichtlen PR, Hugenholtz P, Rafflenbeul W, Jost S, Hecker H. Retardation of the progression of coronary artery disease with nifedipine: results of INTACT. *Circulation*. 1989;80:11–382.
3. Chesbro JH, Webster MWI, Smith HC, et al. Antiplatelet therapy in coronary disease progression reduced infarction and new lesion formation. *Circulation*. 1989;80:11–266.
4. Pearson TA. Coronary arteriography in the study of the epidemiology of coronary artery disease. *Epidemiol Rev*. 1984;6:140–166.
5. McLaughlin PR, Berman ND, Morton BC, Schwartz L, Morch JE. Long-term angiographic assessment of the influence of coronary risk factors on native coronary circulation and saphenous vein aortocoronary grafts. *Am Heart J*. 1977;93:327–333.
6. Nikkila EA, Viikinkoski P, Valle M, Frick MH. Prevention of progression of coronary atherosclerosis by treatment of hyperlipidaemia: a seven-year prospective angiographic study. *Br Med J [Clin Res]*. 1984; 289(6439):220–223.
7. Kuo PT, Hayase K, Kostis JB, Moreyra AE. Use of combined diet and colestipol in long-term (7–7 1/2 years) treatment of patients with type II hyperlipoproteinemia. *Circulation*. 1979;59:199–211.
8. Nash DT, Gensini G, Esente P. Effect of lipid-lowering therapy on the progression of coronary atherosclerosis assessed by scheduled repetitive coronary arteriography. *Int J Cardiol*. 1982;2:43–55.
9. Rafflenbeul W, Smith LR, Rogers WJ, Mantle JA, Rackley CE, Russell RO Jr. Quantitative coronary arteriography: coronary anatomy of patients with unstable angina pectoris reexamined 1 year after optimal medical therapy. *Am J Cardiol*. 1979; 43:699–707.
10. Cohn K, Sakai FJ, Langston MF Jr. Effect of clofibrate on progression of coronary disease: a prospective angiographic study in man. *Am Heart J*. 1975;89:591–598.
11. Bemis CE, Gorlin R, Kemp HG, Herman MV. Progression of coronary artery disease: a clinical arteriographic study. *Circulation*. 1973;47:455–464.
12. Brensike JF, Levy RI, Kelsey SF, et al. Effects of therapy with cholestyramine on progression of coronary arteriosclerosis: results of the NHLBI Type II Coronary Intervention Study. *Circulation*. 1984; 69:313–324.
13. Kimbiris D, Lavine P, Van Den Broek H, Najmi M, Likoff W. Devolutionary pattern of coronary atherosclerosis in patients with angina pectoris: coronary arteriographic studies. *Am J Cardiol*. 1974;33:7–11.
14. Raichlen JS, Healy B, Achuff SC, Pearson TA. Importance of risk factors in the angiographic progression of coronary artery disease. *Am J Cardiol*. 1986; 57:66–70.
15. Bruschke AV, Wijers TS, Kolsters W, Landmann J. The anatomic evolution of coronary artery disease demonstrated by coronary arteriography in 256 nonoperated patients. *Circulation*. 1981;63:527–536.
16. Henderson RR, Rowe GG. The progression of coronary atherosclerotic disease as assessed by cine-coronary arteriography. *Am Heart J*. 1973;86:165–172.
17. Arntzenius AC, Kromhout D, Barth JD, et al. Diet, lipoproteins, and the progression of coronary atherosclerosis: the Leiden Intervention Trial. *N Engl J Med*. 1985; 313:805–811.
18. Marchandise B, Bourassa MG, Chaitman BR, Lesperance J. Angiographic evaluation of the natural history of normal coronary arteries and mild coronary atherosclerosis. *Am J Cardiol*. 1978;41:216–220.
19. Shea S, Sciacca RR, Esser P, Han J, Nichols AB. Progression of coronary atherosclerotic disease assessed by cinevideodensitometry: relation to clinical risk factors. *J Am Coll Cardiol*. 1986;8:1325–1331.
20. Moise A, Theroux P, Taeymans Y, et al. Clinical and angiographic factors associated with progression of coronary artery disease. *J Am Coll Cardiol*. 1984;3:659–667.
21. Blankenhorn DH, Johnson RL, Mack WJ, El Zein HA, Vailas LI. The influence of diet on the appearance of new lesions in human coronary arteries. *JAMA*. 1990; 263:1646–1652.
22. Blankenhorn DH, Johnson RL, Nessim SA, Azen SP, Sanmarco ME, Selzer RH. The Cholesterol Lowering Atherosclerosis Study (CLAS): design, methods, and baseline results. *Controlled Clin Trials*. 1987; 8:354–387.
23. Lipid Research Clinics Program. *The Manual of Laboratory Operations: Lipid and Lipoprotein Analysis*. Washington, DC; 1974. US Dept of Health, Education, and Welfare Publication NIH 75-628.
24. Friedewald WT, Levy RI, Frederickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*. 1972;18:499–502.
25. Breslow NE, Day NE. Unconditional logistic regression for large strata. Chapter 6. In: *Statistical Methods in Cancer Research. Vol. 1. The Analysis of Case-Control Studies*. Lyon, France: International Agency for Research in Cancer; 1980. 192–246.
26. Deupree RH, Fields RI, McMahan CA, Strong JP. Atherosclerotic lesions and coronary heart disease: key relationships in necropsied cases. *Lab Invest*. 1973; 28:252–262.
27. Keys A, Menotti A, Karvonen MJ, et al. The diet and 15-year death rate in the seven countries study. *Am J Epidemiol*. 1986; 124:903–915.
28. Kannel WB, Gordon T. The search for an optimum serum cholesterol. *Lancet*. 1982; 2:374–375.
29. Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: risk factor changes and mortality results. *JAMA*. 1982;248:1465–1477.

A Randomized Trial of an Interim Methadone Maintenance Clinic

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ABSTRACT

Background. Interim methadone maintenance has been proposed as a method of providing clinically effective services to heroin addicts waiting for treatment in standard comprehensive methadone maintenance programs.

Methods. A clinic that provided initial medical evaluation, methadone medication, and AIDS education, but did not include formal drug abuse counseling or other social support services was established in New York City. A sample of 301 volunteer subjects recruited from the waiting list for treatment in the Beth Israel methadone program were randomly assigned to immediate entry into the interim clinic or a control group.

Results. There were no differences in initial levels of illicit drug use across the experimental and control groups. One-month urinalysis follow-up data showed a significant reduction in heroin use in the experimental group (from 63% positive at intake to 29% positive) with no change in the control group (62% to 60% positive). No significant change was observed in cocaine urinalyses (approximately 70% positive for both groups at intake and follow-up). A higher percentage of the experimental group were in treatment at 16-month follow-up (72% vs 56%).

Conclusions. Limited services interim methadone maintenance can reduce heroin use among persons awaiting entry into comprehensive treatment and increase the percentage entering treatment. (*Am J Public Health.* 1991;81:1185–1191)

Introduction

Expansion of drug abuse treatment programs has been one of the more frequently recommended means for controlling the spread of the human immunodeficiency virus (HIV) and AIDS among intravenous drug users (IVDUs). In the United States, the Presidential Commission on the HIV Epidemic[1] and the National Academy of Sciences[2,3] have both advocated "treatment on demand" (providing immediate voluntary entry into treatment) for IVDUs. The National Institute on Drug Abuse has determined that providing treatment for IVDUs will be its primary strategy for reducing the spread of HIV among drug injectors.[4]

The Food and Drug Administration (FDA) has proposed changing the regulations governing methadone maintenance treatment in the United States to permit interim methadone treatment. The term "interim" refers to the provision of limited services to patients awaiting treatment positions in comprehensive methadone programs. Interim clinics would provide intake physical examinations, education about AIDS, and methadone medication to prevent narcotic withdrawal symptoms and to block the euphoric effects of heroin. The methadone would be dispensed daily by a nurse so that there would be frequent contact between the patient and the medical staff and minimal opportunity for diversion of the medication. Interim clinics would not be required to provide ongoing drug abuse counseling, vocational rehabilitation, or the other social services that are incorporated in current federal regulations governing methadone maintenance treatment and that are integral components of most drug-free programs.

According to their proponents, interim clinics would not replace regular methadone maintenance programs, but would provide limited services to heroin addicts who would otherwise be on waiting lists and receiving no drug abuse treatment. The limited services are expected at least to reduce heroin use and the AIDS risks associated with heroin use. Opponents argue that interim methadone maintenance would not address the multiple social and vocational needs of most heroin addicts, and that implementation of limited service treatment might impede expansion of more comprehensive treatment programs, or, in a time of limited public resources for drug abuse treatment, even replace more comprehensive treatment.[1] (For an extended discussion of the proposed FDA regulations that would have permitted interim methadone maintenance treatment see the transcript of hearings conducted by the Select Committee on Narcotics Abuse and Control of the U.S. House of Representatives, March 23, 1990.)

The intensity of debate notwithstanding, there are relatively few data on the effectiveness of interim methadone clinics. Hong Kong and The Netherlands both have some methadone maintenance pro-

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*Harold L. Trigg has died since this study was completed.

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This paper was submitted to the journal June 13, 1990, and accepted with revisions February 20, 1991.

Editor's Note: Also see related Editorial on page 1111 of this issue.

grams that provide few supportive services beyond basic dispensing of the medication. These programs are generally considered effective within those societies,[5,6] but the effectiveness of interim methadone maintenance might be different in the United States, due to cultural differences with respect to drug use and the higher percentage of polydrug use among US heroin addicts. We report here on a random assignment evaluation of an interim methadone clinic in New York City. Subjects receiving interim clinic services were compared to waiting list controls. Change in heroin use was the primary outcome measure utilized; changes in cocaine use and entry into conventional treatment were also examined.

Methods

Interim Clinic Services

The interim clinic was opened in February 1987. It was staffed by a one-quarter-time physician, one full-time registered nurse, one clinic coordinator, one outreach worker, and one full-time research interviewer. The services provided within the clinic included a standard physical examination upon admission and methadone administered by a nurse 5 days per week. Saturday medication and a single take-home dose were provided at another site in the same building. AIDS education, with free distribution of condoms, was also provided at the interim clinic. Urine samples were collected biweekly for toxicologic analyses, which included thin layer chromatography with confirmation by gas chromatography/mass spectrometry performed by Damon Clinical Laboratories of New York.

Results of the urinalysis were known to the treatment staff, but not incorporated into any treatment plan or discussed in formal counseling sessions, as they generally would be in a comprehensive methadone treatment program. Minimal counseling was available on an ad hoc, informal basis, and other supportive service needs could be addressed only by referral to community agencies. The physician could identify medical problems but, except for the most routine of interventions, had to refer patients elsewhere for treatment.

The initial dose of methadone was set by the examining physician based on the examination and drug use history and was typically 20 to 30 mg/day. This initial dose was then increased according to an escalation schedule preset by the physician, generally by 10 mg every third day until a maintenance dosage of approximately 80 mg/day was reached. The nurse was authorized to modify the rate of dose escalation or to stop the escalation process at a stabilization dose other than the scheduled maximum. Dosage adjustment decisions generally reflected subjective perceptions of the patients and instances of missed or vomited medication.

Subjects and Data Collection

Subjects were recruited from the waiting lists of the Beth Israel methadone maintenance program. Separate waiting lists are maintained for the 23 Beth Israel clinics, and there is substantial variation in the time between application for treatment, placement on a waiting list, and admission to one of the regular clinics. At the time of the interim clinic study, the average time spent on a waiting list was approximately 3 months. Participation in the study would be from the time of enrollment until an opening occurred at the clinic to which the subject had originally applied. At that time the subject would be enrolled in the regular clinic and cease participation in the interim clinic study. Participation in the study would neither decrease nor increase the time spent waiting for an opening in a regular clinic.

A written informed consent for participation in the study was obtained from each subject. The project was approved by Beth Israel's Institutional Review Board and the FDA (Investigational New Drug number 28 232). A questionnaire covering demographics, drug use history, AIDS risk behavior, and knowledge of AIDS was administered by a trained interviewer. The subjects were informed that medical and drug use information collected at the interim clinic would not be revealed to the comprehensive clinic to which they would ultimately transfer. Subjects were paid $35 for the completion of intake data collection. A follow-up questionnaire, focusing on drug use since the preceding interview, was administered every 2 weeks.

Residual serum from the blood sample collected as part of the intake examination was stored at −70 °C for later HIV testing. Since the object of the study was to examine the effects of a methadone treatment program that did not include formal counseling, the actual HIV counseling and testing were postponed until completion of the evaluation study. (Relationships between serostatus and drug use behavior will be presented elsewhere.)

After completion of the initial data collection, subjects were assigned randomly to experimental treatment or control conditions. The random assignment was done by administrative staff at a different location. The intake interviewers frequently asked to have individuals placed in the methadone treatment group based on data obtained in the intake interview, but, consistent with the protocol, these requests were not honored. Subjects in the control condition were paid $20 for follow-up interviews. Data were analyzed using the PRODAS system of statistical programs.[7]

Change in Protocol

For the first 3 months after the study inception, there were three experimental conditions: the experimental treatment group immediately received interim clinic treatment as described above and a biweekly follow-up interview; the frequent contact control group received the biweekly follow-up interview, free condoms, and had biweekly urine samples collected; and the minimal contact control group were not reinterviewed until the end of their participation in the study.

Subject recruitment under these conditions was initially rapid, but then slowed dramatically. Discussions with recruited subjects and potential subjects applying for methadone treatment at Beth Israel indicated that the major difficulty was the perception of a low probability (one chance in three) of receiving methadone treatment in the interim clinic study.

In August 1987, the protocol was revised to increase subject recruitment. The minimal contact control group was eliminated from the study, and the time spent in the frequent contact control group was limited to 1 month, after which control subjects were switched into the experimental group and received methadone medication. After this modification, there was no difficulty in recruiting new subjects for random assignment into experimental and control conditions and the 150-person limit on patients receiving methadone in the interim clinic was soon reached. Data from the discontinued minimal contact control group are not included in this report, but are available from the senior author (S.R.Y.).

Data Collection Limitations

One research staff member was designated to conduct baseline and biweekly research interviews for the experimental and control subjects. The outreach worker, who was bilingual (Spanish/English), occasionally assisted in interviewing. It is thus unlikely that the subjects maintained a clear separation between clinic treatment staff and research staff, which may have contributed to underreporting of drug use as presented below.

Given the number of subjects and the severe limitation of clinic space and personnel, some urine collections and interviews were not conducted at the required intervals, especially in the first few months of the study when new intakes and biweekly follow-up interviews for both experimental and control subjects were needed. As the study progressed and the control participants reached their 30th day and were transferred to the experimental treatment group, the total number of study participants stabilized at the maximum of 150 patients receiving methadone, and the rates of missed urines and interviews diminished considerably.

TABLE 1—Selected Demographic and Drug Use Characteristics for Subjects at Entry Into Study (N = 301)

	Experimental Treatment Group (n = 149)		Frequent Contact Group (n = 152)			
Characteristics	N	(%)	N	(%)	χ^2 or t value	*P* Value
Sex						
Male	121	(81)	118	(78)		
Female	28	(19)	34	(22)	0.59	.44
Race						
White	15	(10)	16	(11)		
Black	44	(30)	61	(40)		
Hispanic	89	(60)	75	(49)	3.93	.14
Currently employed	22	(15)	21	(14)	0.06	.85
Living with sexual partner	59	(40)	62	(41)	0.04	.83
Prior drug treatment	115	(78)	122	(81)	0.43	.51
Prior MMTP treatment[a]	89	(60)	81	(53)	1.27	.26
HIV status						
Positive	65	(50)	93	(64)		
Negative	66	(50)	52	(36)	5.93	<.05
Average age at baseline (yr)	34.8		34.4		t=−0.51	.61
Average age began IV drug use (yr)	18.6		19.4		t=1.15	.25
Average age began IV drug use on regular basis (yr)	19.5		20.2		t=1.02	.31

[a] MMTP = methadone maintenance treatment program.

Results

Table 1 shows demographic characteristics and drug use histories for the 301 subjects who were assigned to the experimental and frequent (biweekly) contact control conditions. Ninety-eight percent reported that they had injected drugs at some time in their lives; 92% reported injecting in the month prior to entry into the study, with a mean of over three injections per day. There were no significant differences between groups in demographics or drug use histories. More than half of those who were tested for HIV were seropositive, with a higher percentage seropositive in the control group.

Of the 301 subjects initially enrolled, no follow-up data were obtained from 41 subjects (20 from the experimental group, 21 from the control group). Of these 41 subjects, 32 were lost to contact, 8 were admitted to traditional drug treatment, and 1 was incarcerated before follow-up data could be collected.

Since control subjects were shifted into the experimental treatment after 1 month, primary outcome comparisons between the two groups must be restricted to behavior within that time period. Complete baseline and 1-month follow-up data are available for 169 subjects initially assigned to experimental and frequent contact control groups. The 1-month period was operationally defined as between 15 and 44 days after intake. For subjects with multiple follow-up interviews and urine samples during their participation in the study, the interview and urine sample collected nearest 30 days was used, with the restriction that data from the control subjects reflected only time spent in the control condition. Table 2 presents demographic and drug use history data for these subjects. They do not differ significantly on any of these variables from the total groups assigned to experimental treatment and frequent contact control conditions.

Table 3 presents baseline self-reported drug use and urinalysis results at enrollment for the 169 subjects with complete baseline and follow-up data. There is a general agreement between the self-reports and the urinalysis results. None of the differences between the two groups are statistically significant by chi-square tests.

Table 4 presents self-reported drug use and urinalysis results at the 1-month follow-up. There is clearly substantial underreporting among members of the experimental treatment group. Factors associated with discrepancies between self-reported drug use and urinalysis results will be examined in a separate paper. The drug use analyses presented in this report will be restricted to urinalysis results only. Comparisons of the urinalysis results across experimental treatment and control groups at 1 month using chi-square tests show less heroin use (χ^2 = 15.35, $P < .001$) and more methadone use (χ^2 = 52.86, $P < .001$) in the experimental group. The very small difference in cocaine use was not significant (χ^2 = .09, P = .7) between the two groups.

Comparisons from intake to the 1-month time period within each group showed significantly reduced heroin use (63% to 29%, McNemar χ^2 = 16.45, $P < .001$) and significantly increased methadone use in the experimental group (33% to 92%, McNemar χ^2 = 36.98, $P < .001$). The decrease in cocaine use in the experimental treatment group was not significant (from 77% to 66%, McNemar χ^2 = 1.33, $P < .3$). In the control group, neither the heroin nor cocaine use percentages changed significantly, but there was also a trend toward increased (illicit) methadone use (26% to 37%, McNemar χ^2 = 3.225, $P < .10$).

To determine whether the 169 subjects included in Table 4 were a biased sample of all subjects in the study, we examined urinalysis results for 129 subjects originally assigned to the experimental treatment group and 121 subjects originally assigned to the frequent contact group for whom there was at least one follow-up urinalysis result. These represent 87% of all subjects originally assigned to the experimental treatment group and 80% of all subjects originally assigned to

TABLE 2—Selected Demographic and Drug Use Characteristics for Subjects with 1-month Follow-Up Data (N = 169)

	Experimental Treatment Group (n = 75)		Frequent Contact Group (n = 94)			
Characteristics	N	(%)	N	(%)	χ^2 or t Value	P Value
Sex						
Male	60	(80)	73	(78)		
Female	15	(20)	21	(22)	0.14	.71
Race						
White	11	(15)	6	(6)		
Black	19	(25)	41	(44)		
Hispanic	45	(60)	47	(50)	7.54	<.05
Currently employed	14	(19)	15	(16)	0.19	.66
Living with sexual partner	31	(41)	41	(44)	0.09	.76
Prior drug treatment	60	(80)	78	(83)	0.25	.62
Prior MMTP treatment[a]	50	(67)	51	(54)	2.67	.10
HIV status						
Positive	24	(36)	59	(67)		
Negative	43	(64)	29	(33)	14.91	<.001
Average age at baseline (yr)	33.9		35.7		t=1.71	.09
Average age began IV drug use (yr)	18.3		19.7		t=1.55	.12
Average age began IV drug use on regular basis (yr)	18.8		20.8		t=1.95	.05

[a] MMTP = methadone maintenance treatment program.

TABLE 3—Urinalysis and Self-reported Drug Use at Baseline for Subjects with 1-month Follow-up Data (N = 169)

	Experimental Treatment Group (n = 75)		Frequent Contact Group (n = 94)				
Drugs	n	(%)	n	(%)	χ^2	P Value	Odds Ratio (95% CI[a])
Heroin metabolites							
Urinalysis	47	(63)	58	(62)	0.02	.90	0.960 (0.513–1.795)
Heroin							
Self-report	68	(91)	89	(95)	1.02	.31	
Methadone, nonprescription							
Urinalysis	25	(33)	24	(26)	1.23	.27	0.686 (0.352–1.337)
Self-report	40	(53)	53	(56)	0.16	.70	
Heroin metabolites and/or methadone							
Urinalysis	56	(75)	66	(70)	0.41	.52	0.800 (0.404–1.583)
Heroin and/or nonprescription methadone							
Self-report	72	(96)	91	(97)	0.08	.77	
Cocaine							
Urinalysis	58	(77)	67	(71)	0.79	.37	0.727 (0.361–1.467)
Self-report	63	(84)	83	(88)	0.65	.42	

[a] CI = confidence interval.

the frequent contact control group. The urine sample collected closest to 30 days in the study was used for this analysis. For the experimental treatment group, the urinalysis at follow-up showed 47 (36%) had used heroin, 99 (77%) had used cocaine, and 117 (91%) had used methadone. For the frequent contact control subjects, 72 (60%) had used heroin, 93 (77%) had used cocaine, and 49 (40%) had used methadone. These results are similar to those reported in Table 4, although the percentage for heroin use in the experimental treatment group and the percentages for cocaine use in both the experimental and frequent contact control groups are modestly but not significantly higher. The difference in heroin metabolites between the experimental treatment group and the frequent contact control group at follow-up is still highly significant ($\chi^2 = 13.22$, $P < .001$).

In order to further explore possible determinants of heroin use at 1 month, univariate analyses (chi-square tests and *t* tests) on the 169 subjects with complete intake and 1-month follow-up data were performed to determine if demographic characteristics, drug history variables, or cocaine use were associated with heroin use at 1 month. Only use of cocaine at 1 month and assignment to the control group were significantly associated with heroin use at 1 month, as presented in Table 5.

Inspection of the data showed similar relationships between heroin use and cocaine use at 1 month for both the experimental treatment group and the control group. Among the 75 subjects in the experimental treatment group, 25% had evidence of both cocaine and heroin use in their urine sample, 4% had evidence of heroin use only, 43% had evidence of cocaine use only, and 28% had no evidence of either drug ($\chi^2 = 4.82$, $P < .05$). The relationship was slightly weaker among the 94 subjects in the control group: 46% had evidence of both cocaine and heroin use in their urine sample, 14% had evidence of heroin use only, 24% had evidence of cocaine use only, and 16% had no evidence of either drug ($\chi^2 = 2.86$, $P < .09$). Because the urinalysis performed was capable of detecting cocaine use only for the previous several days and was capable of detecting heroin use for the previous week, these results probably underestimate concurrent use of both drugs for both groups.

Multiple logistic regression was used to examine whether treatment group status and the presence of cocaine metabolites in the 1-month urine sample were independent predictors and whether interactions between experimental group status and demographic or behavioral variables were associated with heroin use at 1 month. Such interaction effects would indicate subjects for whom interim clinic treatment might be particularly effective or not effective in reducing heroin use. The final regression equation is presented in Table 5. Both treatment group status and cocaine use at 1 month were independent predictors of heroin use at 1 month, and none of the possible interactions between treatment group and the demographic and behavioral variables were significant.

A final aspect of the experimental treatment versus control group comparisons was to examine the numbers of subjects who had entered conventional drug treatment programs at the end of data collection in June 1988, i.e., 16 months after the program began. As shown in Table 6, of the 301 subjects originally enrolled in the experimental treatment and the frequent contact control groups, 107 (72%) of those in the experimental group had been enrolled in conventional drug treatment by this data vs 85 (56%) of the controls. This difference for entering conventional treatment was statistically significant ($\chi^2 = 8.23, P < .005$).

TABLE 4—Urinalysis and Self-reported Drug Use at 1-Month Follow-up (N = 169)

Drugs	Experimental Treatment Group (n = 75) n	(%)	Frequent Contact Group (n = 94) n	(%)	χ^2	*P* Value	Odds Ratio (95% CI[a])
Heroin metabolites							
Urinalysis	22	(29)	56	(60)	15.35	<.001	3.550 (1.862–6.771)
Heroin							
Self-report	21	(28)	83	(88)	64.08	<.001	
Methadone,							
Urinalysis	69	(92)	35	(37)	52.86	<.001	0.052 (0.020–0.131)
Methadone, nonprescription							
Self-report	1	(1)	37	(39)	34.61	<.001	
Heroin metabolites and/or methadone							
Urinalysis	71	(95)	68	(72)	14.24	<.001	0.147 (0.049–0.444)
Heroin and/or nonprescription methadone							
Self-report	21	(28)	88	(94)	78.44	<.001	
Cocaine							
Urinalysis	51	(68)	66	(70)	0.09	.76	1.109 (0.575–2.138)
Self-report	29	(39)	79	(84)	37.24	<.001	

[a] CI = confidence interval.

Discussion

This study was based on the hypothesis that participation in a rapid intake, limited service methadone maintenance treatment program would reduce illicit drug use and AIDS risk behaviors among persons with a history of heroin addiction. The study attracted a group of subjects with long histories of illicit narcotic use; the great majority had previous drug abuse treatment experience. These subjects are similar in their demographic characteristics to others admitted to the Beth Israel Methadone Maintenance Treatment Program during the same time period and to other persons admitted to New York State–funded methadone treatment programs in New York City during the same time period.[8] The rate of recent cocaine use was high among these subjects, with over 60% showing cocaine metabolites in the urine specimen taken at study entry.

Random assignment studies have been difficult to conduct in the drug abuse treatment field because of difficulties in recruiting subjects into and keeping them in treatment conditions that they do not want to be in. This study experienced similar difficulties when subjects had only a one in three chance of receiving methadone treatment. After the change in the protocol, the random assignment to either immediate limited service methadone treatment or a time-limited frequent contact control group appears to have been successful. There were few difficulties in recruiting subjects with this change and there was only one variable (HIV status) on which the two groups differed. Since the actual testing of the residual serum for HIV antibody was not done until after random assignment and data collection for this study had been completed, there is no way in which the test results could have influenced the randomization, and we assume that this difference between the experimental treatment and control groups was a random effect.

Ethical considerations required that only volunteers be used in this experimental study. Thus it is not possible to ascertain how these subjects might have differed from persons on the waiting list who chose not to participate and simply waited until a regular treatment position was available. Subject recruitment varied with the perceived likelihood of receiving methadone treatment in the interim clinic, suggesting that large numbers of persons on waiting lists would apply for interim treatment if they were certain that they would receive such treatment.

Because of the substantial discrepancies in self-reported drug use at follow-up, only urinalysis results could be used as a measure of follow-up drug use. Such discrepancies are common when clients in treatment suspect that drug use will lead to some form of negative sanctions.[9] Restricting the comparisons to the urinalysis data prevented assessment of the AIDS risk associated with drug injection, but given the long histories of drug injection among these subjects, it is a reasonably safe assumption that a very high percentage of both heroin and cocaine use was by injection.

Based on the urinalysis results, participation in the interim clinic was associated with a substantial decrease in heroin use. Heroin use at 1 month was reduced by approximately half for the experimental treatment group compared to either heroin use at intake or heroin use among the control group at 1 month. Immediate intake into the limited services methadone treatment was also associated with a higher percentage of subjects being enrolled in comprehensive drug treatment. It is not surprising that receiving even limited interim services would facilitate entry into conventional treatment compared to being on a waiting list. Little is known about why many drug users apply for treatment and are placed on waiting lists but do not enter treatment, but at the least they must be considered missed opportunities for reducing illicit drug use and preventing HIV infection.

The regression analysis for possible interactions between experimental group status and other demographic and behav-

TABLE 5—Potential Predictors of Heroin in Urine Sample at the 1-Month Follow-up (n = 169)

Potential Predictors	Heroin in Urine at 30 Days N	(%)	χ^2 or t Value	*P* Value
Group				
Experimental	22	(29)		
Control	56	(60)	15.35	<.001
Sex				
Male	57	(43)		
Female	21	(58)	2.73	.10
Ethnicity				
White	5	(29)		
Black	27	(45)		
Hispanic	46	(50)	2.50	.29
Employment status at baseline				
Employed	11	(38)		
Not employed	67	(48)	1.01	.31
Living with sexual partner at baseline				
Yes	33	(46)		
No	45	(46)	0.005	.94
Previous drug treatment				
Yes	61	(44)		
No	17	(55)	1.15	.28
Previous MMTP treatment[a]				
Yes	43	(43)		
No	35	(51)	1.29	.25
HIV Status				
Positive	39	(47)		
Negative	34	(47)	0.0008	.98
Cocaine use at 30 days				
Yes	62	(53)		
No	16	(31)	7.15	<.01
Average age at baseline (yrs)	34.9		t=.03	.97
Average age began IV drug use (yrs)	19.6		t=1.07	.29
Average age began IV drug use (yrs) on reguler basis	20.4		t=.91	.36

Note. In the final logistic regression equation: beta for the intercept = −0.29, with a χ^2 of 0.78, p = .3774; beta for group status = −1.30, with a χ^2 of 14.89, P = .0001; beta for cocaine in the urine = .99, with a χ^2 of 7.11, P = .008. Variables were dummy coded to 0/1. For group, a value of 1 indicates that the patient was in the treatment group. For presence/absence of drugs, a 1 indicates that the drug was present in the urine at 30 days.

[a] MMTP = methadone maintenance treatment program.

TABLE 6—Status of Subjects at End of Study (N = 301)

Status	Experimental Treatment Group (n=149) N	(%)	Frequent Contact Group (n=152) N	(%)
Lost to Contact	40	(27)	64	(42)
Drug Treatment	107	(72)	85	(56)
Jail	2	(1)	1	(1)
Death	0		2	(1)

ioral characteristics of the subjects did not produce any significant findings. Thus, the present data do not provide evidence for what types of subjects would do comparatively better or worse in interim methadone treatment. Heroin use was more common among interim treatment subjects using cocaine than those not using cocaine. Indeed, it was rare to find a treatment group subject who had evidence of heroin use but not cocaine use in the follow-up urine sample despite the greater time sensitivity for detecting heroin use. Subjects using cocaine may have used heroin simultaneously in "speedball" injections, which produce a drug effect that many users consider preferable to either drug taken alone. Cocaine users might also have taken heroin as self-medication to reduce the dysphoria following cocaine runs. Screening cocaine users out of interim methadone treatment would probably lead to greater reductions in heroin use during treatment, but would not solve the problem of what to do with the large group using both drugs.

The National Academy of Sciences[2,3] and the Presidential Commission on the HIV Epidemic[1] have recommended large-scale expansion of drug treatment in the United States as a method of reducing both illicit drug use and new HIV infections. Even if a financial commitment were made to provide large-scale expansion, it would still take a period of years for new sites to be found and for staff to be hired and trained. During this multi-year process, interim forms of treatment could be of substantial benefit as drug users waited for openings in conventional treatment. The present study did not compare interim treatment to conventional treatment, and we would object to using these findings as a rationale for substituting limited interim services for conventional treatment. We do believe, however, that these findings strongly support providing interim services rather than leaving drug users on waiting lists for conventional treatment. ☐

Acknowledgments

This research was supported by Cooperative Agreement No. U62/CCU201072-012 from the Centers for Disease Control, Center for Prevention Services.

The authors wish to acknowledge the invaluable contributions made by the staff of the Interim Clinic research project: Juanita Brown, RN, Hector Maldonado, Ruth Royster. and Anita Townley.

References

1. Executive Office of the President. *Report of the Presidential Commission on the Human Immunodeficiency Virus Epidemic.* Washington, DC: Presidential Commission on the Human Immunodeficiency Virus Epidemic; June 1988.
2. Institute of Medicine/National Academy of Sciences. *Confronting AIDS: Directions for Public Health, Health Care, and Research.* Washington, DC: National Academy Press; 1986.
3. Institute of Medicine/National Academy of Sciences. *Confronting AIDS: Update 1988.* Washington, DC: National Academy Press; 1988.
4. Drug abuse treatment and AIDS prevention. Charles R. Schuster. *The Interna-*

tional Working Group on AIDS and IV Drug Use Newsletter. 1989; 4(2):12–13.

5. Buning EC, Coutinho RA, van Brussel, GHA, van Santen GW, van Zadelhoff AW. Preventing AIDS in drug addicts in Amsterdam. *Lancet* I. 1986;(8495:1435) 21:1435. [Letter].
6. The Action Committee against Narcotics. *Hong Kong Narcotics Report 1989.* Hong Kong: Narcotics Division, Government Secretariat; 1989.
7. Conceptual Software, Inc. *PRODAS Reference Manual.* Houston Tex: Conceptual Software, Inc.; 1985.
8. State of New York. Report issued by New York State Division of Substance Abuse Services. *Third Annual Update to the Division of Substance Abuse Services' Statewide Comprehensive Five-Year Plan for 1984–85 through 1988–89, 1986.*
9. Magura S, Goldsmith D, Casriel C, Goldstein PJ, Lipton DS. The validity of methadone clients' self-reported drug use. *Int J Addict.* 1987;22:727–749.

The Environmental Dental Association Raises Ecological Questions as Consumer Reports Purports Mercury/Silver Fillings "Safe"

The May 1991 issue of *Consumer Reports* notes that each year up to 100 million silver-colored [amalgam] fillings are used to restore decayed teeth. The article reports that almost half of each filling is mercury. The Environmental Dental Association (EDA), a San Diego–based organization consisting of nearly 700 practitioners, maintains that the dental industry has overlooked the environmental issues associated with mercury use.

Over 100 tons of mercury are used per year in the dental industry in the United States alone. Mercury, a known poison, is more toxic than lead or arsenic. Invariably, unused amalgam material and old fillings containing mercury wind up in the hands of unregulated scrap metal dealers or in our landfills. An alarming portion of the silver/mercury filling material goes directly down the drain from dental offices, polluting our lakes, rivers, and other bodies of water. The EDA is currently initiating steps to improve on-site water filtration systems in dental offices, which would prevent further mercury contamination of water supplies.

According to the San Diego County Office of Hazardous Waste Management, the determination of hazardous waste status of dental amalgam is incumbent upon the dentist. Therefore, disposal of dental amalgam is essentially unmonitored. A spokesperson for that department stated that they have had a marked increase in calls concerning this issue, which is likely to result in stricter controls in the future.

Mike Cressler, director of Quicksilver Recycling, one of California's few treatment facilities licensed to accept mercury, recently advised the EDA that the amount of environmental contamination from dental mercury residue is significant. He expressed concern over the lack of general awareness on the part of the dental profession regarding the hazardous nature of dental waste, particularly dental amalgam. Cressler emphasized that dentists need to become more environmentally aware. He projects that the increased incidences of health and environmental liability for dentists who do not comply with environmentally-responsible practices, will likely invoke changes in the industry.

The EDA is also concerned about hazardous indoor air quality within dental offices, posing yet another risk to dental personnel who breath mercury vapor on a regular basis. The Occupational Safety and Health Administration (OSHA) estimates that 10% of all dental offices are severely mercury-contaminated. Most have adequate decontamination systems and fail to enforce the wearing of protective gear.

An FDA advisory committee met on March 15 to evaluate the use of mercury in dentistry. Although there was insufficient evidence to warrant a position that silver/mercury fillings are either safe or unsafe, two leading toxicologists testified that the issue was of legitimate concern. Research protocols to investigate health effects are currently being developed.

According to transcripts from the FDA advisory meeting, one of the world's leading authorities on mercury toxicity, Lars Friber, MD, PhD, said that "dental amalgam, from the strictly toxicological point of view, is an unsuitable dental filling material . . . steps should be taken to use, as far as possible, other material than amalgam." Contrastly, *Consumer Reports* concludes that "all risks have to be measured against the alternatives . . . amalgam fillings are still your best bet."

Although the heated debate rages on, the EDA contends that consumers of dentistry should be informed about the nature of the filling materials used and should be advised of all available options. *Consumer Reports* pointed out that the American Dental Association (ADA) opposes any informed-consent laws requiring dentists to inform patients of the possible hazards of mercury fillings and opposes discussion of alternatives.

According to the article published in *Consumer Reports*, "The question in the amalgam debate is whether the minute amount of mercury vapor thought to emanate from fillings has any health effect at all." EDA president, Joyal W. Taylor, DDS, responded to the comment as follows: "As long as this questions remains unanswered, the public deserves the right to know and the right to choose for themselves. Furthermore, we will continue to advocate environmentally responsibly dentistry."

The EDA provides scientific research data, referrals to over 700 mercury-free dentists and doctors throughout the United States and as a public service offers free information packets. The toll-free number for the EDA is 1-800-388-8124.

Public Health Briefs

Aging and the Fitness of Fire Fighters: The Complex Issues Involved in Abolishing Mandatory Retirement Ages

Kurt Saupe, MS, Mark Sothmann, PhD, and Deborah Jasenof, MS

ABSTRACT

This study presents an assessment of the cardiorespiratory health and physical fitness of fire fighters of different ages with the aim of addressing several questions currently being debated on a national level. These include how fire fighters differ across age groups in their health and physical fitness, and what would be the impact on the existing work force of implementing minimum fitness standards as an alternative to mandatory retirement ages.

Fire fighters aged 20-65 years were found to be similar in their cardiorespiratory health and physical fitness to the sedentary segment of the general population of the same age. Particularly worrisome, however, is the low maximal aerobic capacity (31, 28, 26 ml/kg/min), high percent body fat (26, 29, 30), and high resting blood pressure (136/86, 140/90, 143/93 mm Hg) observed in the 40-45-, 50-55-, and 60-65-year-old fire fighters, respectively. In fact, 66%, 83%, and 93% of the fire fighters in these three age groups, respectively, fall below the lowest published recommendations for maximal aerobic capacity in this profession.

These data underscore the need to establish minimum health and physical performance standards for fire fighters and demonstrate the profound impact such minimum standards will have on the existing work force. (*Am J Public Health*. 1991;81:1192–1194)

Introduction

The feasibility of abolishing mandatory retirement ages in public safety occupations is being studied under US congressional mandate. One suggestion has been to replace mandatory retirement ages with minimum standards for health and physical fitness.[1] Before serious discussion regarding developing and implementing standards can begin, the impact of those standards on the current work force must be known. Therefore, we conducted a descriptive case study of a large municipal fire department (approximately 5000 uniformed personnel) in order to (1) describe differences across age groups in selected measures of physical fitness and health using a cross-sectional design and (2) develop hypotheses on the impact of implementing physical performance standards on the existing work force

Methods

Subjects were recruited from a randomized listing of all uniformed full-time fire fighters from a large municipal fire department. The 150 subjects were divided by age into five groups of 30 each: aged 20 to 25, 30 to 35, 40 to 45, 50 to 55, and 60 to 65 years. No more than 40 individuals in each age group had to be contacted in order to obtain the 150 volunteers.

Cardiovascular status was determined from a graded exercise test employing a modified Balke treadmill protocol. ECG, blood pressure, and heart rate were taken at rest; at the end of each stage of exercise; and at 1, 3, and 5 minutes during recovery. Expired gases were collected during the last 1 to 4 minutes prior to volitional fatigue or symptom-limited test termination. Maximal oxygen uptake (VO2max) was measured using an open circuit system as described elsewhere.[2]

Pulmonary function testing was performed using a Collins Survey Spirometer. From a forced vital capacity (FVC) maneuver, forced expiratory volume at 1 second (FEV1) and forced expiratory flow rate between 25% and 75% of FVC (FEF25–75) were calculated using standard procedures.

Percentage of body fat was estimated based upon skinfold thickness of the abdomen, chest, and thigh as measured with a Lange skinfold caliper. The average of three measurements at each site was used in a regression equation to predict percentage of body fat.[3]

Measurements of grip strength, standing long-jump, and situps were made for inclusion with selected cardiovascular and anthropometric variables in the composite fitness evaluation system developed by Davis et al.[4]

Analysis of variance using a post hoc Scheffe test was employed to determine any statistically significant ($P < .05$) differences between the age groups.

Results

Aerobic capacity significantly decreased from the 20- to 25-year-old group (47.7 ml/kg/min) to the 30- to 35-year-old group (37.9 ml/kg/min) and from the 30- to

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This paper was submitted to the journal July 30, 1990, and accepted with revisions February 4, 1991.

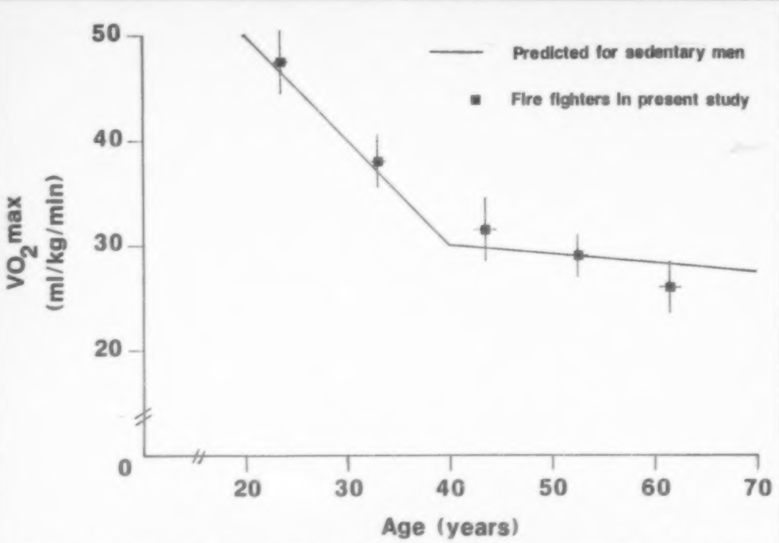

FIGURE 1—Decline in VO2max with age in fire fighters compared with the decline predicted for sedentary individuals by Buskirk and Hodgson.[17]

35-year-old group to the 40- to 45-year-old group (31.5 ml/kg/min) (Figure 1). Given these values for VO2max, Table 1 indicates the percentage of fire fighters in each of the five age groups who would pass various published recommendations for VO2max in fire fighters.

Resting data on blood pressure and pulmonary function are presented for each of the age groups in Table 2. Both systolic and diastolic pressures are significantly higher in the 50- to 55-year-olds and 60- to 65-year-olds than in the 20- to 25-year-olds. FVC was significantly less in the 60- to 65-year-olds than in the 20- to 25-year-old group. Also presented in Table 2 are self-reported data on smoking history and physical activity.

Figure 2 presents lean body weight and fat weight across age groups. Percentage of fat increased significantly from ages 20 to 25 years (8.3%) to 30 to 35 years (19.4%) and again from 30 to 35 years to 40 to 45 years (27.2%).

Table 3 indicates the percentage of fire fighters in each age group exceeding certain clinical criteria. Fire fighters with over 25% body fat are considered to be moderately obese.[5] Individuals with a resting diastolic pressure (DBP) between 90 and 114 mm Hg or a systolic pressure (SBP) greater than 140 mm Hg are considered to the borderline to moderately hypertensive[6] and are categorized as such in Table 3. The percentage of fire fighters in each age group manifesting positive stress tests according to criteria established by the American College of Sports Medicine[7] are listed in Table 3. Exercise hypertension (SBP > 250 or DBP > 120) was the most frequently observed problem in fire fighters aged 40 to 55. In con-

TABLE 1—Percentage of Fire Fighters above Proposed Minimum Values for VO2max

	Age Groups[a]					
Proposed Values	20–25	30–35	40–45	50–55	60–65	Reference
3.0 l/min	83	73	27	33	3	13,14
42 ml/kg/min	87	20	3	0	0	15
39 ml/kg/min	93	40	13	3	3	16
33.5 ml/kg/min	100	83	33	17	7	5

[a]n = 30 for each group.

TABLE 2—Characteristics of Fire Fighters across Age Groups[a]

	Age Groups[a]				
Characteristics	20–25	30–35	40–45	50–55	60–65
Resting systolic blood pressure (mm Hg)[b]	126 ± 10	133 ± 7	137 ± 14	141 ± 15	142 ± 15
Resting diastolic blood pressure (mm Hg)[b]	72 ± 12	82 ± 9	86 ± 9	88 ± 11	88 ± 7
Forced vital capacity (l BTPS)[b]	4.99 ± 0.5	5.30 ± 0.6	4.76 ± 0.7	4.53 ± 0.6	3.80 ± 0.6
FEVl (l BTPS)[b]	4.19 ± 0.4	4.26 ± 0.5	3.70 ± 0.7	3.62 ± 0.5	2.82 ± 0.7
FEF25–75 (l/s)[b]	4.69 ± 0.8	4.43 ± 1.0	3.47 ± 1.1	3.80 ± 1.1	2.32 ± 1.1
% Smokers	10	33	33	27	10
% Previous smokers	10	27	43	50	80
% Never smoked	80	40	23	23	10
% Who exercise more than twice per week	60	40	17	10	33

Note: BTPS = body temperature and pressure, saturated; FEV1 = forced expiratory volume at 1 second; FEF25–75 (l/s) = forced expiratory flow rate between 25% and 75% of forced vital capacity.
[a]n = 30 for each group.
[b]Data are expressed as mean ± standard deviation.

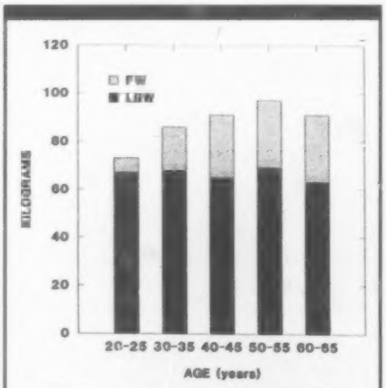

FIGURE 2—Lean body weight (LBW) and fat weight (FW) as a portion of total body weight in fire fighters across age groups.

TABLE 3—Percentage of Fire Fighters in Each Age Group Exceeding Selected Clinical Criteria

	Age Groups[a]				
Criteria	20–25	30–35	40–45	50–55	60–65
> 25% Body Fat	0	27	60	83	77
Borderline to moderate hypertension	20	33	53	70	73
Positive exercise stress test	0	0	7	10	40
Mild to moderate pulmonary impairment	10	13	30	23	53

[a] n = 30 for each group

trast, 10 of the 12 positive stress test results in the 60- to 65-year-old age group were due to ECG abnormalities. The average VO2max of the fire fighters with positive stress tests was 24.5 ml/kg/min. Individuals with either an FEV1/FVC of less than .74 or an FVC less than 79% of predicted[8] are considered to have mild to moderate pulmonary impairment[9] and are also presented in Table 3.

A test battery for assessing the overall physical fitness of fire fighters, which includes measures of strength, cardiovascular status, and pulmonary function and has been shown to be predictive of performance time on a standardized fire suppression test,[4] was also used. According to this rating system, 38% of fire fighters aged 40 to 45, 60% aged 50 to 55, and 96% aged 60 to 65 would be considered poor in their overall fitness for fire fighting.

When fire fighters in each age group were divided into those who reported exercising more than twice a week and those exercising less than twice a week, no significant differences in VO2max, percentage of body fat, or FVC were observed.

Discussion

This case study of a large metropolitan fire department illustrates the complexity of the mandatory retirement age issue faced by municipalities. These data suggest that substantial age-associated declines do occur in the health and physical fitness of fire fighters, although these declines are not necessarily age-dependent. Although fitness standards have been advocated as an alternative to mandatory retirement ages, the data presented here demonstrate that using published recommendations as standards[2,10,13] for the aerobic fitness of fire fighters would have an "adverse impact"[14] on a large segment of the existing work force. Given the existing legal controversies pertaining to adverse impact, a municipality would have to present considerable justification for any proposed standard, which would involve costly research and litigation.[15,16] Lowering standards to avoid adverse impact is legitimate only if public safety is not compromised. Our data suggest that any consideration of a shift in national policy away from mandatory retirement ages should be accompanied by an in-depth examination of the feasibility of developing and implementing medical and fitness standards at the municipal level, and clarification of the controversial issue of whether fitness standards that have adverse impact on certain protected groups are necessary to maintain public safety. □

Acknowledgments

This research was funded by the City of Chicago under contract 65832. The authors wish to thank Clinical Cardiology Group LTD, Chicago Ill, for their essential contribution to this study.

References

1. Davis P, Dotson C. Job performance testing: an alternative to age discrimination. *Med Sci Sport Exer.* 1987;19:179–185.
2. Sothmann M, Saupe K, et al. Advancing age and the cardiorespiratory stress of fire suppression: determining a minimum standard for aerobic fitness. *Human Performance.* 1990;3(4):217–236.
3. Jackson A, Pollock M. Generalized equations for predicting body density of men. *Br. J Nutr.* 1979;40:497–504.
4. Davis P, Dotson C, Santa Maria D. Relationship between simulated firefighting tasks and physical performance measures. *Med Sci Sport Exer.* 1982;14:65–71.
5. Buskirk E. Obesity. In: Skinner J, ed. *Exercise Testing and Exercise Prescription for Special Cases.* Philadelphia, Pa: Lea and Febiger; 1987:149–173.
6. *The 1984 Report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure.* Washington, DC: US Dept of Health and Human Services; 1984.
7. American College of Sports Medicine. *Guidelines for Exercise Testing and Prescription.* Philadelphia, Pa: Lea and Febiger; 1986.
8. Knudson R, Lebowitz M, Holberg C, Burrows B. Changes in the normal maximal expiratory flow-volume curve with growth and aging. *Am Rev Resp Dis.* 1983;127:725–734.
9. American Thoracic Society. *Evaluation of impairment/disability secondary to respiratory disorders.* Medical Section of the American Lung Association; 1986.
10. Zylberstein, M. Fysisk belastning under rokdykning arbetarskyddsstyrelsen, Stockholm (Uppdragerapport nr 488/72). Abstract from Kilbom A. *Scand J Work Environ Health.* 1973;6:48–57.
11. Louhevaara V, Tuomi T, Smolander J, Korhonen O, Tossanainen A, Jaakkola J. Cardiorespiratory strain in jobs that require respiratory protection. *Int Arch Occup Environ Health.* 1985;55:195–206.
12. Davis P, Dotson C. Heart rate responses to firefighting activities. *Ambulatory Electrocardiol.* 1978;1:15–18.
13. Lemon P, Hermiston R. Physiological profile of professional firefighters. *J Occup Med.* 1977;19:558–562.
14. Uniform guidelines on employee selection procedures. *Federal Register.* 1978;43: 38290–38315.
15. Shephard R. Human rights and the older worker: changes in work capacity with age. *Med Sci Sport Exer.* 1987;19:168–173.
16. Faley R, Kleiman L, Lengnick-Hall M: Age discrimination and personnel psychology: a review and synthesis of the legal literature with implications for future research. *Personnel Psych.* 1984;37:327–350.
17. Buskirk E, Hodgson J. Age and aerobic power: the rate of change in men and women. *Fed Proc.* 1987;46:1824–1829.

Malnutrition in Elderly Ambulatory Medical Patients

Aaron Manson, MD, and Steven Shea, MD

ABSTRACT

Elderly ambulatory persons may be especially susceptible to malnutrition, particularly those who are poor and socially isolated or have comorbid chronic medical diseases. We found that 98 of 2,986 persons aged 60 years or older attending a hospital-based medical practice between 1979 and 1989 weighed less than 45.4 kg (100 lbs). All but 1 of these subjects met criteria for malnutrition as judged against age-specific norms for weight. Thus the prevalence of malnutrition in this sample was 3.25% (95% CI 2.61, 3.89%). Interviews and physical examinations of a subsample (n = 16) revealed that all 16 subjects either met anthropometric-based criteria for malnutrition or were being treated for malnutrition. Of the 98 subjects who weighed less than 45.4 kg, 62 (63.3%; 95% CI 53.8, 72.8%) had comorbid conditions that could have contributed to malnutrition. Physicians did not record a diagnosis of malnutrition or weight loss in 47.9% of subjects (95% CI 38.0, 57.8%) and did not prescribe a nutrition supplement for 76.5% of subjects (95% CI 68.1, 84.9%). Subjects treated with nutrition supplement were more likely to have cancer. These findings suggest that malnutrition, both with and without concomitant major comorbid disease, is relatively frequent among elderly ambulatory patients and that a specific nutritional diagnosis is not made in many cases. We suggest that weight under 45.4 kg in an elderly person is a useful criterion for identifying elderly patients at nutritional risk. (*Am J Public Health.* 1991;81:1195–1197)

Introduction

In 1968 Jean Mayer testified before Congress that as many as 10% of Americans were malnourished due to poverty.[1] During the ensuing decade, government programs reduced malnutrition, but more recently funding for these programs has diminished.[2] The two largest nutritional surveys in the United States, the Ten-State Nutrition Survey[3] and the National Health and Nutrition Examination Surveys (NHANES I and II),[4–6] collected comprehensive data but were conducted in the 1970s, and neither study estimated the prevalence of malnutrition in the elderly. Other studies of malnutrition or weight loss among the elderly are anecdotal, describe small numbers, include hospitalized patients, or do not estimate the prevalence of malnutrition in the populations studied.[7–24] We therefore studied the prevalence of malnutrition among elderly ambulatory patients.

Methods

The study population consisted of outpatients aged 60 years or older, who were receiving care in the medical faculty practice at Columbia-Presbyterian Medical Center, and whose recorded weights in the practice database were 45.4 kg (100 lbs) or less on at least two office visits during the period from 1979 to 1989. Patients were routinely weighed on a balance scale at most visits. In 1989, there were 8,956 patient records in the database; of these 2,987 (33.3%) referred to patients who were age-eligible; one age-eligible patient did not have a recorded weight in the database. Hospital charts of 124 of the 128 age- and weight-eligible patients were obtained, and these patients' weight of 45.4 kg or less on at least two visits in 98 of these 124 patients was confirmed (Table 1).

We reviewed the medical records of these 98 patients using standardized data forms. The diagnoses of malnutrition or weight loss were considered to have been recorded if there was explicit mention in the chart subsequent to the index visit. Treatment of weight loss or malnutrition was considered to have been provided if chart notes recorded the prescription of liquid nutrition supplement. Alcoholism, cancer, severe chronic pulmonary disease, congestive heart failure, dementia, depression, or stroke were noted as comorbid conditions that could contribute to malnutrition.

We further defined nutritional status on the basis of two sets of age- and sex-specific norms, NHANES[25] and the Ten-State Nutrition Survey.[3] Subjects were considered severely malnourished if measurements of weight for height[25] or weight[3] were below the fifth percentile according to these norms. Subjects were considered moderately malnourished if these measurements were between the fifth and fifteenth percentiles.

Results

Laboratory findings in the 98 subjects were consistent with although not specific for malnutrition (Table 1). Of the 48 subjects with recorded heights and weights, 28 (58.3%) were severely malnourished, and 15 (31.3%) were moderately malnourished as measured against the norms from NHANES. All five of the remaining subjects, who could not be classified because their heights were below the minimum height in these distributions, either were taking nutrition supplements or had a diagnosis of malnutrition in the medical record. Measured against norms based on weight alone[3] 2 subjects more than 85 years old could not be classified, 48 (49.0%) were severely malnourished, 42 (42.9%) were moderately malnourished, and 6 (6.1%) were above the 15th percentile. Of the 6 subjects above the 15th percentile, 4 were described as malnourished

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This paper was submitted to the Journal August 7, 1990, and accepted with revisions February 20, 1991.

TABLE 1—Characteristics of 98 Subjects with Weights of 45.4 kg (100 lbs) or Less Attending a General Medicine Ambulatory Care Practice in New York City, 1979–1989[a]

	Mean	SD
Age (years)	75.2	± 9.8
Follow-up time (months)	33.1	± 32.4
Weight (kg)	40.9	± 3.9 (range 26.1–45.4)
Height (cm)	155.4	± 10.1 (range 134.6–175.3)
Albumin (mg/dl)	4.0	± 0.46
Hemoglobin (mg/dl)	12.3	± 1.8
Absolute lymphocyte count (cells/microliter)	1,647	± 761

[a]Five (5.1%) of the subjects were men, and 28 (28.6%) were receiving Medicaid.

TABLE 2—Characteristics of 98 Subjects with Weights of 45.4 kg (100 lbs) or Less Classified by Nutritional Diagnosis as Recorded in the Medical Record

	Subjects with Diagnosis of Malnutrition or Weight Loss (n = 51)		Subjects with No Nutritional Diagnosis (n = 47)	
Basic Characteristics	Mean	SD	Mean	SD
Age (years)	75.6	± 9.4	74.7	± 10.4
Follow-up time (months)	29.9	± 27.4	36.5	± 37.0
Weight (kg)[a]	40.8	± 4.0	41.1	± 3.9
Height (cm)	153.3	± 11.2	157.8	± 8.2
Albumin (mg/dl)[b]	3.97	± 0.42	4.04	± 0.51
Hemoglobin (mg/dl)[c]	12.2	± 1.6	12.4	± 2.0
Absolute lymphocyte count[d] (cells/microliter)	1,767	± 816	1,519	± 690
Number (percent) men	4 (7.8%)		1 (2.1%)	
Comorbid Disease[e]	Subjects (Percent) with Diagnosis Malnutrition or Weight Loss (n = 51)		Subjects (Percent) with No Nutritional Diagnosis (n = 47)	
Alcoholism	5 (9.8%)		3 (6.4%)	
Cancer	11 (21.6%)		4 (8.5%)	
Chronic pulmonary disease	8 (15.7%)		15 (31.9%)	
Depression	10 (19.6%)		9 (19.1%)	
Congestive heart failure	9 (17.6%)		11 (23.4%)	
Dementia	9 (17.6%)		5 (10.6%)	
Stroke	3 (5.9%)		5 (10.6%)	

[a]Height was available for 48 subjects.
[b]Serum albumin was available for 80 subjects.
[c]Hemoglobin was available for 77 subjects.
[d]Absolute lymphocyte count was available for 60 subjects.
[e]Sixty-two subjects had one or more comorbid conditions; 28 subjects had more than one.

in the medical record, and a fifth subject met criteria for malnutrition as measured against norms of weight for height.[25] Both extremely elderly subjects were described as malnourished in the medical record. Thus the false positive rate in classifying these 98 subjects using weight alone was 1% or less. We therefore estimated the prevalence of malnutrition among subjects over the age of 60 years in this medical practice as 97 of 2,986 (3.25%; 95% CI 2.61, 3.89%).

The diagnosis of malnutrition was recorded in the charts of 23 subjects (23.5%), and the diagnosis of weight loss was recorded in 28 additional subjects (28.6%, Table 2). Twenty-three subjects (23.5%) were treated with calorie supplement (Table 3). Thus 47 of 98 subjects (47.9%; 95% CI 38.0, 57.8%) had no recorded diagnosis of malnutrition or weight loss, and 75 (76.5%; 95% CI 68.1, 84.9%) were not treated with calorie supplement.

Comorbid conditions that could have contributed to malnutrition were present in 62 subjects (63.3%; 95% CI 53.8, 72.8%, Table 2). Presence of a comorbid condition was not significantly associated with a recorded diagnosis of malnutrition or weight loss; however, such subjects were more likely to have cancer (odds ratio = 2.96; 95% CI 0.87, 10.04).

A substudy (N = 16) of subjects who made scheduled visits to the practice between July 1989 and February 1990 found that the observed weight was 45.4 kg or less in all subjects and that the mean observed weight differed from that in the chart by less than 1%. Using norms for bone-free upper arm muscle area based on triceps skinfold and mid-arm circumference measurements,[25] we found 11 subjects to be below the fifteenth percentile. Of the remaining 5, 4 had a recorded diagnosis of malnutrition or were taking nutrition supplement. The remaining subject met criteria for malnutrition according to norms for weight[3] and weight for height.[25] Thus, all 16 substudy subjects either met anthropometric criteria for malnutrition or were being treated for malnutrition, including 7 subjects with no recorded nutritional diagnosis.

Discussion

This study found that 3.25% of ambulatory elderly patients in a medical practice had evidence of malnutrition, that weight of 45.4 kg (100 lbs) or less is a useful screening criterion for malnutrition, and that the diagnosis of malnutrition was noted in only 52.1% of the 98 malnourished subjects. Of the 98 subjects, 62 (63.3%) had an underlying disease that probably contributed to malnutrition, but more than a third did not; this suggests that malnutrition among the elderly is often present in the absence of major comorbid illness.

It is likely that our method underestimated the prevalence of malnutrition. A weight of 45.4 kg may be too low to identify some malnourished men with larger frames. Thus, only 5 subjects in this series were male.

Although data were collected retrospectively, it is unlikely that information regarding other diagnoses was incomplete since there were computer-based lists of medical diagnoses for all subjects, and the mean observation time in the medical record was 33 months. Weights recorded in the chart were measured during routine care; however, weights measured in the validation substudy corroborated those in the chart.

Although their findings are not conclusive, several longitudinal studies have found that extreme leanness is associated with adverse medical outcomes.[26,27] Al-

TABLE 3—Characteristics of 98 Subjects[a] with Weights of 45.4 kg (100 lbs) or Less Classified by Nutritional Treatment as Recorded in the Medical Record[b]

	Treated (n = 23)		Not Treated (n = 75)	
Men	Mean	SD	Mean	SD
Age (years)	75.0	± 8.7	75.2	± 10.2
Follow-up time (months)*	18.7	± 19.7	37.4	± 34.5
Weight (kg)	39.9	± 4.1	41.2	± 3.8
Height (cm)	154.3	± 11.1	155.9	± 9.6
Albumin (mg/dl)	3.80	± 0.41	4.07	± 0.46
Hemoglobin (mg/dl)**	12.5	± 1.0	12.2	± 2.0
Absolute lymphocyte count (cells/microliter)	1,403	± 567	1,720	± 802

[a] Four (17.4%) of the treated subjects were men, and 7 (30.4%) had cancer.
[b] Fisher's Exact Test was used to determine statistical significance.
*P = .002. **P = .003.

though the prognostic significance of a weight of 45.4 kg or less (100 lbs) in the elderly is not known, our data indicate that this easily remembered indicator is a useful screening criterion for malnutrition in the elderly and can be used to define the prevalence of this condition.[28] ☐

Acknowledgments

We wish to thank Dr. Haydee Rondon for help with interviewing and examining patients, Dr. Robert Sciacca for help in performing statistical analysis, and the physicians of the Associates in Internal Medicine for providing patients for this study.

References

1. Mayer J. *Human Nutrition: Its Physiological, Medical and Social Aspects: A series of eighty-two essays.* Springfield, Ill: CC Thomas; 1972:635.
2. Physician Task Force on Hunger in America. *Hunger in America. The Growing Epidemic.* Middletown, Conn: Wesleyan University Press; 1985:8–10, 175–176.
3. *Ten-State Nutrition Survey, 1968–1970. III—Anthropometry, Highlights.* Atlanta: Centers for Disease Control; 1972. US Dept of Health, Education, and Welfare publication DHEW (HSM) 72-8134.
4. Abraham S, Lowenstein FW, O'Connell OE. *Preliminary Findings of the First Health and Nutrition Examination Survey, United States 1971–1972; Anthropometric and Clinical Findings.* Washington, DC: Public Health Service; 1975. US Dept of Health, Education, and Welfare publication DHEW 75–1229.
5. *Dietary Intake Source Data, United States 1971–1974.* Hyattsville, Md: National Center for Health Statistics; 1979. US Dept of Health, Education, and Welfare publication (PHS) 79-1221.
6. *Dietary Intake Source Data, United States 1976–1980. Vital and Health Statistics, Series 11, No. 231.* Hyattsville, MD: National Center for Health Statistics; 1983. US Dept of Health, Education, and Welfare publication DHHS (PHS) 83-1681.
7. Ohlson MA, Jackson L, Boek J, et al. Nutrition and dietary habits of aging women. *Am J Pub Health.* 1950;40:1101–1108.
8. Gillum HL, Morgan AF. Nutritional status of the aging. *J Nutr.* 1955;55:265–288, 289–303, 449–468, 655–670.
9. Lyons JS, Trulson MF. Food practices of older people living at home. *J Gerontol.* 1956;11:66–72.
10. Taylor GF. A clinical survey of elderly people from a nutritional standpoint. In: Exton-Smith AN, Scott DL, eds. *Vitamins in the Elderly.* Bristol, England: John Wright & Sons; 1968:51–56.
11. Kohrs MB, O'Neal R, Preston A, Eklund D, Abrahams O. Nutritional status of elderly residents in Missouri. *Am J Clin Nutr.* 1978;31:2186–2197.
12. Yearick ES. Nutritional status of the elderly: anthropometric and clinical findings. *J Gerontol.* 1978;33:657–662.
13. Yearick ES, Wang ML, Pisias SJ. Nutritional status of the elderly: dietary and biochemical findings. *J Gerontol.*. 1980; 35:663–671.
14. O'Hanlon P, Kohrs MB. Dietary studies of older Americans. *Am J Clin Nutr.* 1978; 31:1257–1269.
15. Clarke RP, Schlenker ED, Merrow SB. Nutrient intake, adiposity, plasma total cholesterol, and blood pressure of rural participants in the (Vermont) Nutrition Program for Older Americans (Title III). *Am J Clin Nutr.* 1981;34:1743–1751.
16. Koh ET, Chi MS. Clinical signs found in association with nutritional deficiencies as related to race, sex, and age for adults. *Am J Clin Nutr.* 1981;34:1562–1568.
17. Garry PJ, Goodwin JS, Hunt WC, Hooper EM, Leonard AG. Nutritional status in a healthy elderly population: dietary and supplemental intakes. *Am J Clin Nutr.* 1982; 36:319–331.
18. Bowman BB, Rosenberg IH. Assessment of the nutritional status of the elderly. *Am J Clin Nutr.* 1982;35:1142–1151.
19. Bistrian BR, Blackburn GL, Vitale J, Cochran D, Naylor J. Prevalence of malnutrition in general medical patients. *JAMA.* 1976;235:1567–1570.
20. Bistrian BR, Blackburn GL, Hallowell E, Heddle R. Protein status of general surgical patients. *JAMA.* 1974;230:858–860.
21. Steffee WP. Malnutrition in hospitalized patients. *JAMA.* 1980;244:2630–2635.
22. Marton KI, Sox HC, Krupp JR. Involuntary weight loss: diagnostic and prognostic significance. *Ann Intern Med.* 1981;95:568–574.
23. Rabinovitz M, Pitlik SD, Leifer M, Garty M, Rosenfeld JB. Unintentional weight loss: a retrospective analysis of 154 cases. *Arch Intern Med.* 1986;146:186–187.
24. Kergoat M, Leclerc BS, Petitclerc C, Imbach A. Discriminant biochemical markers for evaluating the nutritional status of elderly patients in long-term care. *Am J Clin Nutr.* 1987;46:849–861.
25. Frisancho AR. New standards of weight and body composition by frame size and height for assessment of nutritional status of adults and the elderly. *Am J Clin Nutr.* 1984;40:808–819.
26. Andres R. Mortality and obesity: the rationale for age-specific and height-weight tables. In: Andres R, Bierman EL, Hazzard WR, eds. *Principles of Geriatric Medicine.* New York: McGraw Hill;1985:311–318.
27. Waaler HT. Height, weight and mortality. The Norwegian experience. *Acta Med Scand Suppl.* 1984;679:3–56.
28. Brody JA. Toward quantifying the health of the elderly. *Am J Pub Health.* 1989; 79:685. Editorial.

Suicides among Older United States Residents: Epidemiologic Characteristics and Trends

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ABSTRACT

Suicide rates for elderly US residents decreased between 1950 and 1980, but have increased recently. We analyzed suicide mortality trends using national mortality data for the period 1980 through 1986. Suicide rates during this period increased for each 5-year age group over age 65. Elderly White males have the highest suicide rates and experienced a rate increase of 23%. The rate for Black males rose by 42%. Divorced males have the highest age-adjusted sex- and marital status-specific rates, and experienced a rate increase of 38% over the 7-year period. Suicide rates among older US residents vary by region of the country and are highest in the West. Rates increased in all regions except the Northeast. Firearms are the most common method of suicide in the elderly, and firearm use increased during this period from 60% to 66% of all suicides. Given the recent increase in suicide rates for the elderly and the magnitude of the problem in this age group, it is again important to direct our attention to the problem of suicide in the elderly and recognize the need for effective prevention strategies. (*Am J Public Health.* 1991;81:1198–1200)

Introduction

Suicide rates for elderly US residents decreased between 1950 and 1980,[1] but have increased recently.[2] Previous studies of suicide among persons of all ages have identified high risk groups including whites, males, widowed or divorced persons, and residents of the western United States.[1,3,4] In this article we examine recent trends in suicide rates for elderly US residents, with an analysis of risk groups and methods used in suicide.

Methods

Data on suicide deaths for the years 1980 through 1986 are from annual mortality data tapes compiled by the National Center for Health Statistics (NCHS), Centers for Disease Control. Suicides analyzed in this article are deaths among persons over age 65 with external cause-of-death codes E950–E959, according to the International Classification of Diseases, Ninth Revision (ICD-9).

Population data used in the calculation of rates by age, sex, and race are from annual computer data tapes produced by the US Bureau of the Census. Marital status-specific and region-specific rates were calculated using population estimates from Bureau of the Census publications.[5,6] Marital status-specific rates were age-adjusted by the direct method (10-year age groups), using the 1980 population as the reference. Because of the small numbers of suicides among other racial groups, we limited our analysis of race-specific rates to the Black and White races.

Results

From 1980 through 1986, 36 789 suicides were reported among US residents over age 65 years, and crude annual suicide rates increased for this age group by 21% (from 17.8 to 21.5 per 100 000). Rate increases ranged from 9% (from 16.2 to 17.7 per 100 000) for the 65- through 69-year age group to 38% (from 18.0 to 24.8 per 100 000) for the 80- through 84-year age group (Table 1). During this period, men accounted for 80% of the suicides among persons over age 65, and rates for men increased for each 5-year age group over age 65.

White males had the highest race- and sex-specific suicide rates for persons over age 65 years (Table 2), and from 1980 to 1986 their rate increased 23% (from 37.2 to 45.6 per 100 000). Suicide rates for Black men increased 42% (from 11.4 to 16.2 per 100 000), and the suicide rates for White women increased 17% (from 6.4 to 7.5 per 100 000).

The 1986 suicide rate for divorced males over age 65 was 3.2 times the rate for married men and 18.9 times the rate for married women (Table 3). From 1980 to 1986, the rate for divorced men increased 38% (from 79.5 to 109.7 per 100 000). Rates for women increased, regardless of the marital status category. However, because the numbers are small, the rates fluctuate more.

In 1986, the western United States had the highest suicide rate for persons over age 65 (29.5 per 100 000), the Northeast had the lowest rate (13.6 per 100 000), and the southern and northcentral regions had intermediate rates (23.7 and 20.2 per 100 000, respectively). From 1980 to 1986, the suicide rate decreased slightly in the Northeast, whereas rates increased in the northcentral (22%), western (23%), and southern (29%) regions.

From 1980 to 1986 firearms were the most common method of suicide for both men and women over age 65 years (Figure 1). Over the 7 years, 73% of older men and 29% of older women committed suicide with firearms. Firearm use increased from

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This paper was submitted to the journal July 9, 1990, and accepted with revisions February 20, 1991.

60% of all over-65 suicides in 1980 to 66% in 1986 for both sexes combined.

Discussion

Suicide is the third leading cause of deaths from injury among older US residents, following deaths from falls and from motor vehicle crashes.[2] From 1980 through 1986, suicide rates increased for each 5-year age group over age 65; the only other age groups that experienced a rate increase of at least 5% over this period were the 60- through 64-year age group (10%) and the 15- through 19-year age group (20%) (NCHS Detailed Mortality Tapes 1980–1986).

Although the demographic groups at high risk for suicide among the elderly are similar to those for all ages combined,[1,3,4] differences in suicide rates between demographic subgroups have become more pronounced. For example, the rate ratio between men and women in the >85 age group increased from 9.2 to 13, and the rate ratio for the western region compared with the northeast region increased from 1.6 to 2.2. It is noteworthy, however, that from 1980 to 1986 the rate ratio for Whites over age 65 compared with Blacks narrowed, due to the larger increase in suicide rates among Black men.

In 1986, 66% of suicides among the elderly were committed with a firearm, compared with 57% among persons under age 65. Methods of suicide differ more between men and women over age 65 than for other age groups. In 1986, 75% of male victims over age 65 died of gunshot wounds, compared with 61% of male victims under age 65. During the same year, 31% of female victims over age 65 died of gunshot wounds, compared with 41% under age 65 (NCHS Detailed Mortality Tapes 1986). The use of drugs and poisons is a common method of suicide for older women. Not only do methods of suicide have important implications for the care of acutely suicidal persons, but the availability of specific methods as risk factors for suicide merits further study.

TABLE 1—Numbers of Suicides and Suicide Rates by Sex and Age Group for US Residents over Age 65, 1980–1986ª

		Age Group									
		65–69		70–74		75–79		80–84		>85	
Year	Sex	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
1980	Male	1098	28.1	955	33.2	766	41.3	446	43.3	345	49.9
	Female	324	6.6	253	6.4	175	5.9	90	4.6	85	5.4
	Total	1422	16.2	1208	17.7	941	19.5	536	18.0	430	18.9
1981	Male	1033	26.0	925	31.4	742	38.8	484	46.2	354	50.4
	Female	356	7.2	260	6.4	168	5.5	93	4.7	63	3.8
	Total	1389	15.6	1185	16.9	910	18.4	577	19.1	417	17.7
1982	Male	1129	26.0	1053	34.9	859	43.7	518	48.3	363	50.4
	Female	361	7.2	269	6.5	197	6.3	104	5.2	66	3.8
	Total	1490	16.5	1322	18.5	1056	20.8	622	20.1	429	17.5
1983	Male	1139	27.7	1108	35.9	913	45.2	622	56.7	397	53.7
	Female	370	7.3	307	7.3	210	6.6	126	6.1	92	5.1
	Total	1509	16.4	1415	19.4	1123	21.5	748	23.6	489	19.3
1984	Male	1212	29.1	1239	39.3	939	45.2	614	54.7	390	51.6
	Female	387	7.6	303	7.1	208	6.3	133	6.3	92	4.9
	Total	1599	17.2	1542	20.7	1147	21.4	747	23.0	482	18.4
1985	Male	1236	29.1	1252	39.0	1035	48.5	714	61.9	428	55.4
	Female	345	6.7	312	7.2	247	7.4	130	6.0	89	4.6
	Total	1581	16.8	1564	20.7	1282	23.3	844	25.4	517	19.1
1986	Male	1344	30.7	1368	41.9	1161	52.8	695	58.5	484	61.1
	Female	363	6.9	339	7.7	272	7.9	155	6.9	94	4.7
	Total	1707	17.7	1707	22.3	1433	25.5	850	24.8	578	20.7

ªPer 100 000 population per year.

TABLE 2—Numbers of Suicides and Suicide Rates by Race for US Residents over Age 65, 1980–1986ª

	White				Black			
	Male		Female		Male		Female	
Year	No.	Rate	No.	Rate	No.	Rate	No.	Rate
1980	3490	37.2	896	6.4	97	11.4	18	1.4
1981	3407	35.7	899	6.3	105	12.1	30	2.3
1982	3786	38.8	961	6.6	108	12.2	23	1.8
1983	4020	40.3	1068	7.2	128	14.1	19	1.4
1984	4233	41.6	1081	7.2	129	14.0	24	1.7
1985	4495	43.2	1059	6.9	143	15.2	38	2.7
1986	4853	45.6	1168	7.5	156	16.2	35	2.4

ªPer 100 000 population per year.

Possible limitations of our data should be discussed. Underreporting has been suggested to be a major source of error in the determination of suicide rates.[4,7] Although the precise extent to which deaths are underreported as suicides is unknown, it is probably a small proportion and should not affect the analysis of trends in suicide rates.[8,9] The denominator used in the calculation of marital status-specific rates is derived from the current population survey, and excludes institutionalized persons. Other rates were calculated using census data and intercensal estimates of the resident US population. Although this methodological difference may affect actual rates, any effect on trends in age-adjusted rates should be minimal.

Known risk factors for suicide that may contribute to high rates and to an increase in rates for the elderly include depression, alcoholism, and chronic illness.[10] Although overall rates of depression may be increasing in the United States,[11] this increase would have to be occurring disproportionately among the elderly to explain the prominent and largely isolated increase in suicide rates for this group. We are not aware of such evidence. Alcohol abuse is less prevalent among the elderly than in younger age groups.[12] It is possible, however, that more people with alcohol-related illnesses are living to an older age, resulting in an increase in the prevalence of alcoholism among persons over age 65 and therefore more at-risk people in this age group. Fi-

TABLE 3—Age-Adjusted Suicide Rates by Sex and Marital Status for US Residents over Age 65, 1980–1986*

	Marital Status							
	Married		Never Married		Divorced		Widowed	
Year	Male	Female	Male	Female	Male	Female	Male	Female
1980	26.9	5.0	61.6	4.9	79.5	10.8	76.5	7.7
1981	27.0	5.3	55.3	5.8	74.7	10.3	69.5	7.3
1982	27.5	5.4	74.6	5.9	99.9	16.7	81.5	7.1
1983	29.9	5.7	69.8	6.8	103.4	13.2	76.5	8.0
1984	31.0	5.6	65.9	7.5	113.6	18.3	81.4	8.2
1985	33.1	5.5	53.5	6.1	101.1	15.0	78.7	8.0
1986	34.5	5.8	53.5	5.9	109.7	18.4	85.9	8.4

*Per 100 000 population per year.

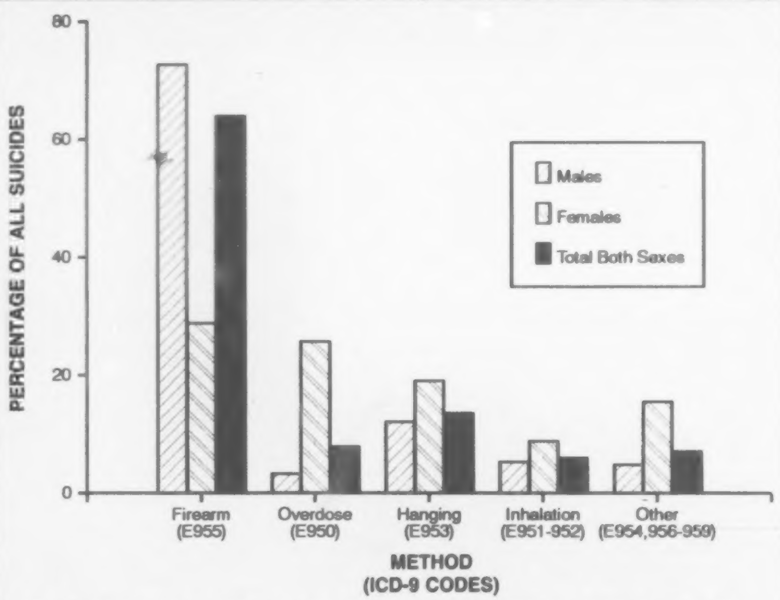

FIGURE 1—Method of suicide used by US residents over age 65 years, 1980–1986

nally, advances in medical care are helping to prolong the lives of persons with chronic illnesses. This could result in higher suicide rates for the elderly, because suicide risk may be higher among chronically ill persons.[13]

Several recommendations can be made based on our findings. We need to consider how current approaches to suicide prevention can better reflect the special circumstances of older persons. Crisis services, for example, may have the greatest impact on the prevention of youth suicide, which may be more impulsive in nature.[14] For older adults, however, addressing the increasingly prevalent problem of social isolation,[15] perhaps reflected in the high suicide rates among divorced persons, may be a more effective prevention strategy. Studies that address the role of economic factors in the precipitation of suicide are needed, as are analytic studies to evaluate, on the individual level, the relationship between correlates of social disruption, such as divorce or geographic migration, and the risk of suicide. Finally, health providers should be increasingly aware of the potential for suicide among the elderly, particularly among those at highest risk, such as divorced older men and Black men. ☐

Acknowledgment

We thank Sharon Clanton for her help with data management.

References

1. Centers for Disease Control. *Suicide Surveillance, 1970–1980.* Atlanta, Ga: CDC, 1985.
2. Sattin RW, Nevitt MC. Injuries in later life: epidemiology and environmental aspects. In: Williams TF, Evans JG, eds. *Oxford Textbook of Geriatric Medicine*, Oxford University Press, in press.
3. Smith JC, Mercy JA, Conn JM. Marital status and the risk of suicide. *Am J Public Health.* 1988;78:78–80.
4. Monk M. Epidemiology of suicide. *Epidemiol Rev.* 1987;9:51–69.
5. US Bureau of the Census. *Current Population Reports.* Series P-20, Nos. 365, 372, 380, 389, 399, 410, 418. Marital status and living arrangements: March. Washington, DC: US Govt Printing Office; 1980–1986.
6. US Bureau of the Census. *Current Population Reports.* Series P-25, No. 1010. State population and household estimates, with age, sex, and components of change: 1981–1986. Washington, DC: US Govt Printing Office; 1987.
7. Hlady WG, Middaugh JP. The underrecording of suicides in state and national records, Alaska, 1983–1984. *Suicide Life Threat Behav.* 1988;18:237–244.
8. O'Carroll PW. A consideration of the validity and reliability of suicide mortality data. *Suicide Life Threat Behav.* 1989; 19:1–16.
9. Kleck G. Miscounting suicides. *Suicide Life Threat Behav.* 1988;18:219–236.
10. Blumenthal SJ. Suicide: a guide to risk factors, assessment, and treatment of suicidal patients. *Med Clin North Am.* 1988; 72:937–971.
11. Klerman GL, Weissman MM. Increasing rates of depression. *JAMA.* 1989; 261: 2229–2235.
12. Gurland BJ, Cross PS. Epidemiology of psychopathology in old age. *Psychiatr Clin North Am.* 1982;5:11–26.
13. Whitlock FA. Suicide and physical illness. In: Roy, MB, ed. *Suicide.* Baltimore, Md: Williams & Wilkins; 1986:151–170.
14. Frances A, Blumenthal S. Personality as a predictor of youthful suicide. In: *Alcohol, Drug Abuse, and Mental Health Administration. Report of the Secretary's Task Force on Youth Suicide.* Volume 2: *Risk Factors for Youth Suicide.* Washington, DC: US Govt Printing Office; 1989:160–171. US Department of Health and Human Services publication (ADM) 89-1622.
15. US Bureau of the Census. *Current Population Reports.* Series P-23, No. 138. Demographic and socioeconomic aspects of aging in the United States. Washington, DC: Govt Printing Office, 1988;85–99.

Development and Validation of a Simple Device to Estimate Birthweight and Screen for Low Birthweight in Developing Countries

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ABSTRACT

Low birthweight (<2500 g) is the major factor associated with the death of infants within the first 4 weeks of life. The mean birthweight of newborn babies of a certain geographic area reflects the quality of maternal and child health care service as well as the degree of socioeconomic development of that particular region. Hence, birthweight is being used increasingly as an indicator for health and socioeconomic planning. However, in a developing country such as Thailand, two-fifths of the babies are delivered at home and are not weighed because scales are not available. To solve this problem in rural areas, a circular nomographic chart was developed with which the birthweight can be computed from a newborn baby's chest and mid-arm circumferences. Preliminary trials comparing these charts with standard baby scales showed a high degree of accuracy with sufficient sensitivity and specificity. (*Am J Public Health*. 1991;81:1201–1205)

Introduction

Low-birthweight (<2500 g) is the major factor associated with the death of infants within the first 4 weeks of life. The mean birthweight of newborn babies of a certain geographic area may reflect the quality of maternal and child health care service as well as the degree of socioeconomic development of that particular region.[1–4] However, in a developing country such as Thailand, two fifths of the babies are delivered outside hospitals and are not weighed because scales are not available.[5] Anthropometric parameters such as chest circumference,[6] mid-arm measurement,[7] and foot length[8] have been used to identify low birthweight and a newborn at risk. The World Health Organization has studied the chest and arm circumferences in correlation with birthweight, aiming to develop chest or arm bands that would indicate that the birthweights are above or below a specified cutoff point (i.e., 2500 g). However, no effort has been made to correlate anthropometric parameters with actual birthweight. This study uses multiple parameters of chest and arm circumferences and the length of a newborn to estimate birthweight and to validate this new method in various centers throughout Thailand.

Methods

The Training Data Set

The training data set undertaken at the Department of Pediatrics, Lerdsin Hospital, from January to March 1985, involved 402 newborn babies, 197 male and 205 female. The duration of pregnancy at the time of delivery ranged from 36 to 40 weeks according to Nägele's formula. The measurements taken within 24 hours of birth included chest circumference at nipple line in centimeters, arm circumference, midway between the acromion process and the tip of elbow in centimeters, crown-to-heel length in centimeters, and weight in grams.

Measuring Instruments

The measuring instruments consisted of a tailor's metric measuring tape, tested against a standard ruler, and a baby metric beam balance (SECA, made in Germany), tested for accuracy with standard weights. All measurements were performed by the same pediatrician throughout the study.

Sample Size

The sample size was calculated after Lachin[9] to estimate the sample size for the correlation (r) .85, setting $\alpha = .05$ and the power of test at .80 (for a two-tailed test).

Data Analysis

Stepwise multiple regression analysis was employed to formulate the best linear prediction equation.[10] The least squares method was used for fitting the "best" straight line to a given set of data in order to relate two variables.

Testing Data Set

The testing data set of body weight and chest and arm circumferences of 3222 newborn infants from eight hospitals in north, northeast, central, and south Thai-

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This paper was submitted to the journal July 23, 1990, and accepted with revisions February 20, 1991.

land was used for analysis. Measurements were taken within 24 hours of birth. The study population was derived from two sets of data. The first set consisted of 1612 newborn babies from the project "Low Birth Weight Newborn Babies of High Risk Mothers" at Chulalongkorn University Hospital, and at regional maternal and child health hospitals in Chiengmai, Nakornsawan, Rajburi, Yala, and Masharaj Hospital at Nakorn-srithamaraj. This was a case-control study, with a 1:2 ratio of low birthweight (LBW) to normal birthweight infants.

The second set of data consisted of 1610 newborn babies from Lerdsin Hospital and the Khon-Kaen University Hospital in Bangkok.

The first set was collected by nurses, using standardized measuring methods, and the second set was obtained by the doctors responsible for the project.

A diagnostic test was employed to reveal sensitivity and specificity of positive predictive values, negative predictive values, and accuracy of the new device in comparison with the gold standard (a baby metric beam balance) in screening for LBW infants.

TABLE 1—Mean ± Standard Deviation for Weight, Chest, and Arm Circumferences, and Body Length of the Study Samples

Measurement	Male (n = 197)	Female (n = 205)	Total (n = 402)
Weight (g)	3079.1 ± 424.0	2941.9 ± 427.4	3009.0 ± 430.7
Chest (cm)	32.8 ± 1.8	32.2 ± 1.8	32.5 ± 1.8
Arm (cm)	10.7 ± 1.0	10.6 ± 1.0	10.6 ± 1.0
Length (cm)	49.5 ± 2.0	48.6 ± 1.9	49.0 ± 2.0

TABLE 2—Stepwise Regression for Birthweight Estimation

Step	Variable Entered	Coefficient	SE[a] of Coefficient	Error[b]	R^2 (%)[c]
1	Chest	207.2	5.4	199.8	78.5
	Constant	−3725.2			
2	Chest	149.6	8.3	183.3	82.0
	Arm	134.4	15.4		
	Constant	−3281.5			
3	Chest	104.8	9.0	167.0	85.1
	Arm	131.5	14.0		
	Length	57.4	6.3		
	Constant	−4607.0			

[a] SE = standard error.
[b] Standard error of estimate.
[c] R^2 = Coefficient of determination.

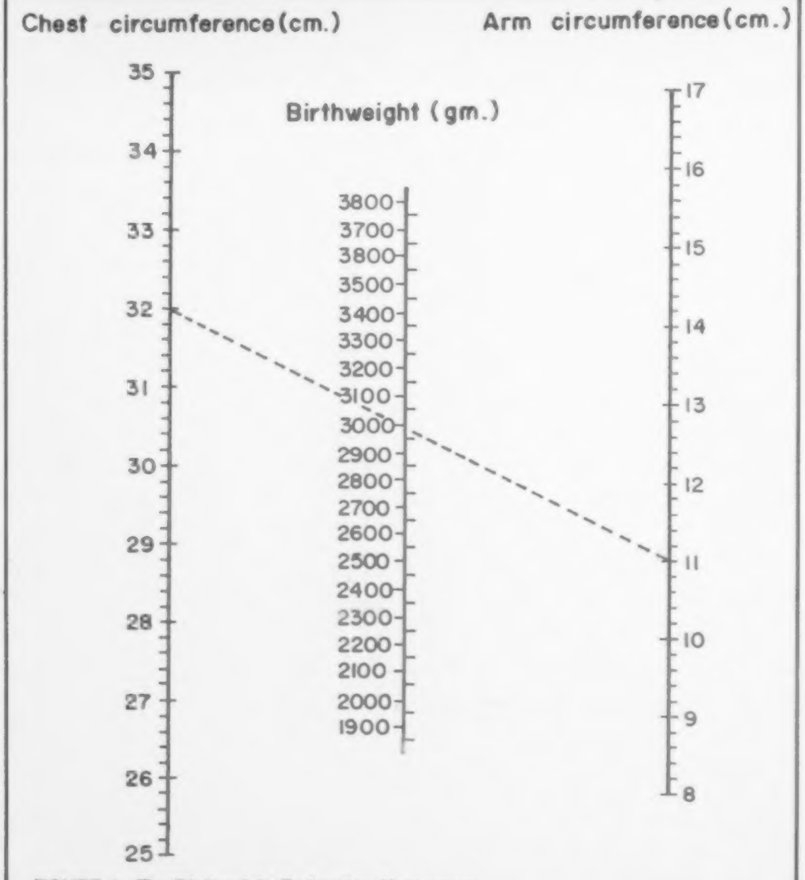

FIGURE 1—The Birthweight Estimation Nomogram

Results

The data from the study of 402 newborn babies are summarized in Table 1. The correlation coefficients (r) of weight versus chest and arm circumferences and body length were 0.89, 0.82, and 0.78, respectively.

Although the combined chest and arm circumferences and length gave the best correlation with birthweight ($r = .92$), measuring the length of a newborn baby is usually impractical and may be subject to relatively large error. We therefore chose the second best equation (Table 2), using the combined chest and arm circumferences to estimate birthweight ($r = 0.90$, $r^2 = 82.0\%$, and standard error (SE) = ±183.3 g). Based on this equation, a nomogram was constructed (Figure 1) and, subsequently, a circular birthweight estimation device was developed (Figure 2).

The mean and standard deviations (SD) of chest and arm circumferences of 3222 newborn babies in various study centers are shown in Table 3. The mean of chest circumferences was about 31 cm, and arm circumferences was about 10 cm.

The differences of means between the weight obtained by standard scales and the estimated weight obtained by the new device (Figure 2) are also shown. The differences appear to be within ±187 g.

The diagnostic values of the new device, when the arbitrary point for picking LBW infants is set at 2500 g, are shown in Table 4. The sensitivity was lowest (56.9%) at Nakornsawan and highest (94.5%) at Chulalongkorn hospital. The specificity was lowest (86.1%) at Khon-Kaen and highest (98.6%) at Yala. The accuracy varied from 83.2% to 95.7%.

If the arbitrary point for defining LBW neonates is set at 2600 g on the new device, the sensitivity at all centers increased, ranging from 71.3% to 97.3%, the specificity ranged from 78.4% to 97.2%, and the accuracy ranged from 79.8% to 92.3%, as shown in Table 5. If the arbitrary point was set at 2700 g, the sensitivity increased to 100% at Chulalongkorn Hospital but the specificity declined to 58.7% at Khon-Kaen Hospital (Table 6).

These results showed that the optimum arbitrary point of all data combined for defining LBW neonates when using the new device should be 2600 g and can be confirmed by the receiver operating characteristic curve shown in Figure 3. The curve became steeper when the arbitrary point was changed from 2500 g to 2600 g, which means that the sensitivity increased markedly while only a small degree of specificity was lost. However, the curve became linear when the arbitrary point was 2700 g, which indicates a very small increase in sensitivity but marked loss of specificity.

Discussion

Birthweight of infants estimated from the Chulalongkorn birthweight estimation device differed by only ±187.4 g from weight obtained by a standard scale. This difference is not clinically significant unless the birthweights are below 2000 g.

The sensitivity and specificity of the new device varied when arbitrary points of LBW infants were set at 2500, 2600, and 2700 g. There was less variation in accuracy, however. The optimum arbitrary point on the new device, as determined by a receiver operating characteristic curve, was found to be 2600 g. The sensitivity increased to 100% when the arbitrary point was 2700 g, but the specificity was low, indicating that there would be an increased number of falsely identified LBW infants.

The Chulalongkorn birthweight estimation device appears promising as an appropriate substitute for obtaining the birthweight of newborns in places where scales are not readily available. While the theoretical error was ±183.3 g, the prac-

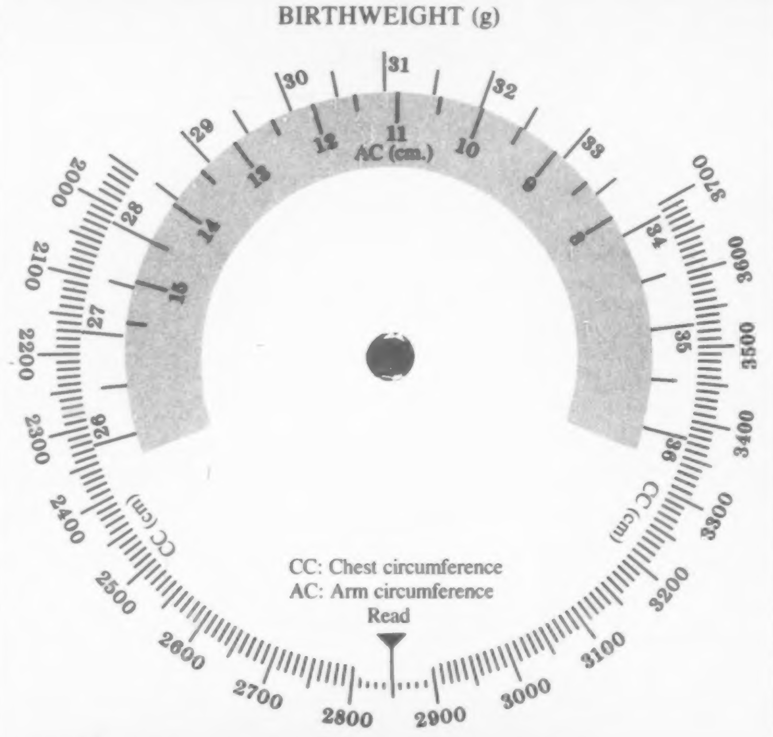

FIGURE 2—Chulalongkorn University Birthweight Estimation Device

TABLE 3—Mean ± Standard Deviation of Arm and Chest Circumferences and Weight of Newborn Babies

Location	n	Chest (cm)	Arm (cm)	Scale Weight	Estimated Weight	Difference of Mean
LS	606	32.3 ± 1.8	9.9 ± 0.9	2996.0 ± 378.7	2854.5 ± 331.4	141.5
KK	1004	31.6 ± 1.6	9.9 ± 0.7	2974.2 ± 360.8	2786.8 ± 321.0	187.4
CU	260	30.9 ± 2.2	9.9 ± 0.9	2826.6 ± 482.7	2668.6 ± 438.7	159.9
CM	339	31.3 ± 2.2	10.0 ± 1.0	2768.6 ± 482.6	2747.4 ± 440.5	21.2
NS	215	31.5 ± 2.3	10.6 ± 1.2	2887.5 ± 532.2	2861.4 ± 501.9	26.1
RB	215	31.4 ± 2.4	10.3 ± 1.2	2779.6 ± 554.8	2790.0 ± 501.5	8.7
YL	300	31.3 ± 2.4	10.8 ± 1.4	2759.2 ± 544.3	2857.1 ± 542.6	−97.9
NT	276	31.8 ± 2.5	10.7 ± 1.2	2797.6 ± 515.3	2925.2 ± 531.3	−127.6

Note. LS = Lerdsin General Hospital; KK = Khon-Kaen University Hospital; CU = Chulalongkorn University Hospital; CM = Chiengmai MCH Hospital; NS = Nakornsawan MCH Hospital; RB = Ratchaburi MCH Hospital; YL = Yala MCH Hospital; NT = Nakorn-srithamaraj MCH Hospital.

TABLE 4—Diagnostic Values for Detecting Low Birthweight Babies (<2500 g) When the Arbitrary Point Is Set at 2500 g on the New Device

Diagnostic Values	LS	KK	CU	CM	NS	RB	YL	NT
Sensitivity	79.4	88.1	94.5	75.5	56.9	65.3	65.8	57.4
Specificity (%)	97.6	86.1	93.0	96.1	96.5	97.5	98.6	98.4
Accuracy (%)	95.7	86.2	93.5	89.4	83.2	86.7	87.4	84.4
+PV (%)	79.4	37.0	—	—	—	—	—	—
−PV (%)	97.6	98.8	—	—	—	—	—	—

Note: +PV, positive predictive value; −PV, negative predictive value; Positive and negative predictive values of case-control study cannot be calculated, since estimates of prevalence of low birthweight were not available.

LS = Lerdsin General Hospital; KK = Khon-Kaen University Hospital; CU = Chulalongkorn University Hospital; CM = Chiengmai MCH Hospital; NS = Nakornsawan MCH Hospital; RB = Ratchaburi MCH Hospital; YL = Yala MCH Hospital; NT = Nakorn-srithamaraj MCH Hospital.

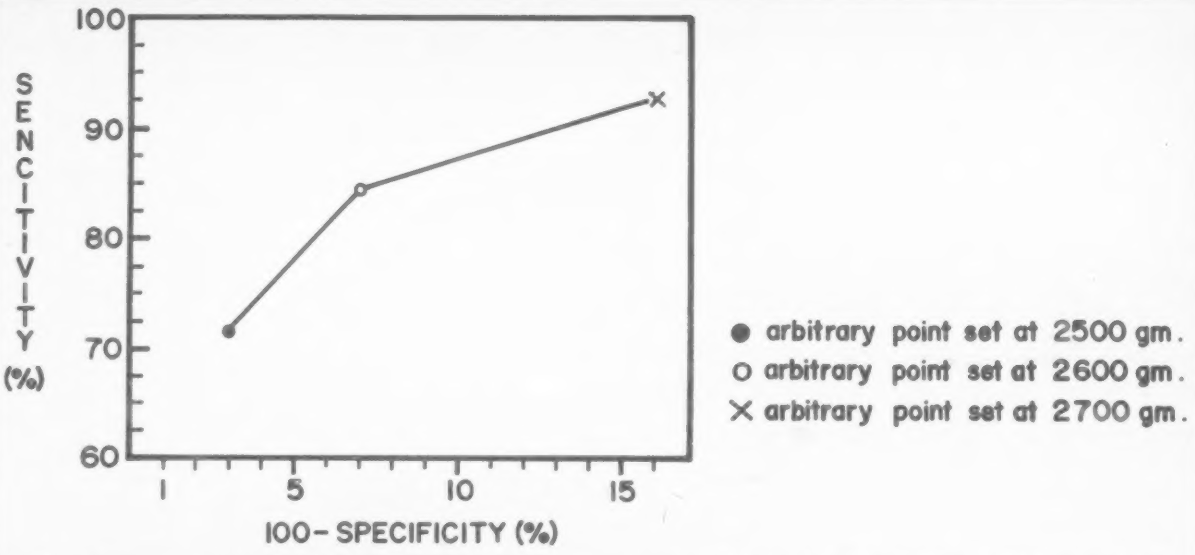

FIGURE 3—Receiver Operating Characteristics Curve of Birthweight Determination in Diagnosing Low Birthweight (All Data Combined, N = 3222)

TABLE 5—Diagnostic Values for Detecting LBW Babies (<2500 g) When the Arbitrary Point is Set at 2600 g on the New Device

Diagnostic Values	LS	KK	CU	CM	NS	RB	YL	NT
Sensitivity (%)	87.3	95.2	97.3	89.5	80.6	76.2	81.6	71.3
Specificity (%)	92.9	78.4	85.6	93.8	93.7	98.5	95.7	97.2
Accuracy (%)	92.3	79.8	88.9	92.3	89.3	89.0	91.0	88.4
+PV (%)	59.1	—	—	—	—	—	—	—
−PV (%)	98.4	—	—	—	—	—	—	—

Note. +PV, positive predictive value; −PV, negative predictive value; Positive and negative predictive values of case-control study cannot be calculated, since estimates of prevalence of low birthweight were not available.

LS = Lerdsin General Hospital; KK = Khon-Kaen University Hospital; CU = Chulalongkorn University Hospital; CM = Chiengmai MCH Hospital; NS = Nakornsawan MCH Hospital; RB = Ratchaburi MCH Hospital; YL = Yala MCH Hospital; NT = Nakorn-srithamaraj MCH Hospital.

TABLE 6—Diagnostic Values for Detection of LBW Babies (<2500 g) When the Arbitrary Point is 2700 g on the New Device.

Diagnostic Values	LS	KK	CU	CM
Sensitivity (%)	92.1	96.6	100.0	93.9
Specificity (%)	80.5	58.7	74.3	81.8
Accuracy (%)	81.7	62.0	81.5	85.8
+PV (%)	35.6	—	—	—
−PV (%)	98.9	—	—	—

Note. +PV, positive predictive value; −PV, negative predictive value; positive and negative predictive values of case-control study cannot be calculated, since estimates of prevalence of low birthweight were not available.

LS = Lerdsin General Hospital; KK = Khon-Kaen University Hospital; CU = Chulalongkorn University Hospital; CM = Chiengmai MCH Hospital.

tical error was ±187.4 g when compared with standard weighing scales.

The diagnostic test revealed the value of the new device. The optimum arbitrary point for screening an LBW infant was 2600 g, at which the sensitivity was 97.3%, the specificity was 97.2%, and the accuracy was 92.3%. This new device is inexpensive and convenient and is a good example of appropriate technology for primary health care. Use of the device should be encouraged throughout developing countries such as Thailand. ☐

Acknowledgments

The authors are grateful to Assistant Professor Supudda Pawanarit of the Department of Mathematics, Faculty of Science, for valuable suggestions concerning the mathematical formulas used in this study. They also wish to thank Dr. Prathan Kanchanalai, Head of the Department of Pediatrics, Lerdsin Hospital, Dr. Dittee Choengcharoen of Siriraj Hospital, and Associate Professor Chitr Sitthi-amorn of Chulalongkorn University for helpful advice. The authors also thank Associate Professor Wongkulapat Snidvongs for assistance in preparing the manuscript.

References

1. Petterson R, Regnstrom N, Sterky G, Taube A. *Birthweight Distribution and Socioeconomic Variables.* Geneva: World Health Organization; 1978. Sarec Report No. R2:45.
2. Mahner J. *Birthweight as a New Development Indicator.* Geneva: World Health Organization; 1978. Sarec Report No. R2:33.
3. Rao MVS. Social-economic indicators for development planning. *Int Soc Sci J.* 1975; 23:132.
4. Panpiamrat K. *Indicator for Poverty at the Umphur level: Concepts and Calculation Method.* [In Thai]. Rural Thailand 2526: Office of National Economic and Social Development Board; 1982.
5. Chaturachinda K, Hiranraks A, Auamkul N, Amornvichet P, Kanchanasinith K, Piy-

apinyo P. *A Nationwide Study of Birthweight in Thailand.* Presented at the Fourth Fertility Research Investigators' Meeting; September 28-29, 1985; Asia Hotel, Pattaya, Thailand.
6. Bhargava SK, Ramji S, Kumar A, Mohan M, Marwah J, Sachder HPS. Mid-arm and chest circumferences at birth as predictors of low birth weight and neonatal mortality in the community. *Br Med J.* 1985; 291:1617–1619.
7. De Vaquera MV, Townsend JW, Arroyo JJ, Lechtig A. The relationship between arm circumference at birth and early mortality. *J Trop Pediatr.* 1983;29:167–174.
8. Daga SR, Daga AS, Patole S, Kadam S, Mukadam Y: Foot length measurement from foot print for identifying newborn at risk. *J Trop Pediatr.* 1988;34:16-19.
9. Lachin JM. Introduction to sample size determination and power analysis for clinical trials. *Controlled Clin Trials.* 1981;2:93–113.
10. Draper NR, Smith H. *Applied Regression Analysis.* New York: Wiley; 1966.

Oral Rehydration Program Evaluation by Quality Assurance Sampling in Rural Haiti

June N. Pierre-Louis, MPH

ABSTRACT

An oral rehydration program in rural Haiti was evaluated by quality assurance sampling. The quality assurance sampling method and its application are described. The indicators measured were knowledge of the oral rehydration salts packet, knowledge of preparation of the solution from the packet, oral rehydration therapy use, and knowledge of preparation of the solution from salt and sugar. Coverages of the first two indicators were adequate, coverages of the latter two were inadequate. The method is a useful low-cost approach to evaluation of program coverage. (*Am J Public Health.* 1991;81:1205–1207)

Introduction

Oral rehydration therapy (ORT) is a cost-effective treatment for diarrheal disease.[1] While the scientific basis for oral rehydration is established, and programs are in place, many programs have not been evaluated at the local level.

In rural Haiti local programs typically target populations of less than 50,000.[2] To meet the need for a low-cost, population-based survey method, quality assurance sampling (QAS), a small sample method,[3] was used to evaluate a local ORT program in Haiti. The QAS method and its application in Haiti are described.

Methods

Study Population

Freres is a rural area located 20 km from Port-au-Prince, the capital of Haiti. The population (13,750 in 1987) lives on dirt roads within one km of a partly paved main road which bisects the area. Freres is targeted by a primary health care program managed by a private hospital in the area. The program maintains a census of the target population.

Promotion of ORT takes place at community meetings and on the radio. Health workers teach mothers to prepare the solution of oral rehydration salts (ORS) from a packet, and from salt and sugar, and to use ORT when their children under three years of age have diarrhea.[2]

Indicators

Widely accepted indicators of program outcome were selected by managers and funders of oral rehydration programs in Haiti and included: ORT use, knowledge of the packet, knowledge of preparation of the solution from the packet, and from salt and sugar.[4,5] The use rate was defined as the proportion of episodes of diarrhea in children under three years of age receiving ORT, whether the solution was prepared from the packet or salt and sugar.[4] The rate of knowledge of preparation of the solution was defined as the proportion of mothers of children under age three knowing the correct quantities of ingredients in a solution prepared from the packet or from salt and sugar.

The survey questionnaire is given in the Appendix.

Quality Assurance Sampling Method

The QAS method was first used for quality control in manufacturing. Product quality was assessed based on the probability of observing a particular proportion of defective items in a sample relative to a null hypothesis stating a proportion of defective products. This probability is determined by the binomial distribution. The goal of the QAS method is hypothesis testing rather than parameter estimation. The major advantage of QAS is the sample size which is small.

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TABLE 1—Hypothesized Proportions of Nonrecipients and Recipients, Null Hypotheses and Sample Sizes, Oral Rehydration Program Evaluation, Quality Assurance Sampling, Freres, Haiti, 1987

Indicator	Hypothesized Proportion of Nonrecipients P_0	Hypothesized Proportion of Recipients/ Short-term Program Goal $1\text{-}P_0$	Null Hypothesis	Sample Size n	Critical Number Nonrecipients d^*
ORT Use	0.95	0.05	$P_0 \geq 0.95$	20	17
Knowledge of Packet	0.30	0.70	$P_0 \geq 0.30$	45	9
Knowledge of Preparation from Packet	0.70	0.30	$P_0 \geq 0.70$	45	27
Knowledge of Preparation from Salt and Sugar	0.90	0.10	$P_0 \geq 0.90$	45	37

TABLE 2—Coverage and Estimated Proportions of Nonrecipients and Recipients, Oral Rehydration Program Evaluation, Quality Assurance Sampling, Freres, Haiti, 1987

Indicator	Sample Number Nonrecipients d	Critical Number Nonrecipients d^*	Coverage	Estimated Proportion of Nonrecipients	Estimated Proportion of Recipients
ORT Use	19	17	Inadequate	≥0.95	<0.05
Knowledge of Packet	8	9	Adequate	<0.30	≥0.70
Knowledge of Preparation of the Solution from Packet	25	27	Adequate	<0.70	≥0.30
Knowledge of Preparation of the Solution from Salt and Sugar	40	37	Inadequate	≥0.90	<0.10

The rate or coverage of use of ORT in the target population, for instance, is determined to be adequate or inadequate based on finding a certain proportion of children under age three not receiving the benefits of ORT in a sample. The null hypothesis is that ORT coverage is inadequate; that is, the proportion of nonrecipients in the sample is greater than some hypothesized proportion of nonrecipients based on short-term program objectives (see Table 1). The null hypothesis is so stated to control the probability of concluding that a community has adequate health coverage when it is actually inadequate. The probability of such a false negative result was set at 0.10, (α). This error is more serious than a false positive error (β) in which the conclusion is that health coverage is inadequate when it is really adequate.

The following expression holds:[3]

$$\alpha = \Pr\{d \leq d^*\}$$

where d is the observed number of nonrecipients in the sample and d^* is the critical number of nonrecipients in a given sample size. When $d \leq d^*$ the null hypothesis that coverage is inadequate is rejected and it is concluded that coverage is adequate.

The critical number of nonrecipients in the sample was calculated by:[3]

$$d^* = nP_0 - z_\alpha [nP_0(1 - P_0)]^{1/2}$$

where n is the sample size; P_0 is the hypothesized proportion of nonrecipients; $1 - P_0$ is the hypothesized proportion of recipients; and z equals 1.28 for α equal to 0.10 using a one-sided test. Fractions were rounded down to the next whole number.

Null hypotheses and values of P_0, $1 - P_0$, n and d^* are given in Table 1 for each indicator.

Sample sizes were obtained from tables developed by Lemeshow, *et al.*,[6] of (n, d^*) pairs for selected values of α, β, and P_0. Values of β are estimates of the difference between true and hypothesized proportions of nonrecipients. A sample size of 45 was chosen for the indicators except ORT use. A mother was asked about ORT use only if she said her child had diarrhea in the past two weeks to minimize recall bias. Sixty-three mothers were interviewed, of which 20 had children under age three who had diarrhea in the past two weeks.

A systematic sample was drawn from the census of the target population maintained by the primary health care program.

The nonresponse rate was 9 percent.

Results

Coverages of ORT use and of knowledge of preparation of the solution from salt and sugar of mothers of children less than three years of age were determined to be inadequate (Table 2). The ORT use rate was estimated to be <0.05. The rate of knowledge of preparation of the solution from salt and sugar was estimated to be <0.10.

Coverages of knowledge of the packet and of preparation of the solution from the packet of mothers of children under age three were determined to be adequate. The rate of knowledge of the packet was estimated to be ≥0.70. The rate of knowledge of preparation of the solution from the packet was estimated to be ≥0.30.

Discussion

The QAS survey in Freres revealed that the primary health program had inadequate coverage of ORT use in the target population despite adequate rates of knowledge of the packet and of preparation of the solution from the packet. The probability that the latter two rates are false negative results is ≤0.10.

The estimated ORT use rate in Freres in 1987 (<0.05) was much lower than the rate for Haiti in 1985 (0.23 of mothers of children less than five).[7] The estimated rate of knowledge of the packet in Freres

(≥0.70) was similar to the national rate in 1985 (0.58).

The QAS evaluation was relatively low-cost because the sample size was small. One interviewer was required for the survey and the evaluation was completed in two days. The QAS method is useful for evaluation of local oral rehydration programs in Haiti. ☐

Acknowledgments

I express my appreciation to Jean-Jacques Lamour for his assistance in the training and supervision of the Freres health workers. This research was supported by a USAID grant to the *Institut Haitien de l'Enfance* and the Haitian Arab Center, Port-au-Prince, Haiti.

References

1. Cash RA: Oral rehydration in the treatment of diarrhea. In: Chen LC, Scrimshaw NS (eds): Diarrhea and Malnutrition: Interaction, Mechanisms and Interventions. New York: Plenum Press, 1983; 203–221.
2. Institut Haitien de l'Enfance: Action pour la survie de l'enfant Haiti. *Port-au-Prince: Editions de l'Enfance,* 1987.
3. Lemeshow S: Sampling techniques for evaluating health parameters in developing countries. Board on Science and Technology for International Development, National Research Council Working Paper. Washington, DC: National Academy Press, 1988.
4. Tulloch J, Burton P: Global access to oral rehydration salts and use of oral rehydration therapy. World Health Stat Q 1987; 40:110–115.
5. Chowdhury AM: Evaluating community ORT programmes: Indicators for use and safety. Health Pol Plann 1986; 1:214–221.
6. Lemeshow S, Hosmer DW, Klar J, Lwanga SK: Adequacy of Sample Size in Health Studies. New York: John Wiley & Sons, 1990; 24–28, 213–214.
7. Ministry of Public Health and Population: Rapport final d'evaluation du program national de controle de la diarrhee et de promotion de l'allaitement maternel. Port-au-Prince: Ministry of Public Health and Population, 1985.

APPENDIX

Survey Questionnaire, Quality Assurance Sampling Survey, Freres, Haiti, 1987.

1. Did (name of the child) have diarrhea in the past two weeks? (If not, pass to question 3).
Yes No

2. What did you give (name of the child)?
(Circle all spontaneous responses of the mother. Do not mention alternatives.)
Home Remedy Doctor's Medicine Packet Solution
Salt and Sugar Solution Nothing

3. Do you recognize this packet? (Show the ORS packet.)
What is it called? ______________

4. How do you prepare the packet ORS solution? (Circle all spontaneous responses of the mother. Do not mention alternatives.)
3 Kola Bottles of Water 1 Rum Bottle of Water 1 ORS Packet
Solution Boiled Other ______________ Doesn't Know

5. How do you prepare the salt and sugar ORS solution? (Circle all spontaneous responses of the mother. Do not mention alternatives.)
2 Large Spoons of Sugar 1 Small Spoon of Salt
1 Rum Bottle of Water 3 Kola Bottles of Water
2 Small Spoons of Sugar 1 Pinch of Salt
1 Kola Bottle of Water Solution Boiled
Other ______________ Doesn't Know

Impaired and Disabled Elderly in the Community

Amasa B. Ford, MD, Ann W. Roy, PhD, Marie R. Haug, PhD, Steven J. Folmar, PhD, and Paul K. Jones, PhD

ABSTRACT

A nine-year representative, longitudinal study of 1,598 urban elderly shows that two to eight times as many impaired or disabled are cared for in the community as in institutions. Younger age, male gender, better income, and living with others, especially children, favor continuing care in the home. (*Am J Public Health.* 1991; 81:1207–1209)

Introduction

The purpose of the present study is to report information about the distribution between community and long-term institutional care of a representative sample of urban elderly over a nine-year period. We identify supporting factors that have enabled from two to eight times as many of the most impaired and disabled to remain in the community as were admitted to long-term nursing home care.

Methods

In 1975 a random sample of 1,598 noninstitutionalized persons age 65 years and older, living in the City of Cleveland, Ohio, were interviewed in their homes using the Duke OARS instrument.[1,2]

Nine years later, in 1984, the vital status of all but 13 individuals (0.8 percent) was ascertained. Among the 805 who were known to have survived, 647 (80.4 percent) were reinterviewed, using the same instrument. Information sufficient to determine institutionalization was ob-

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TABLE 1—Survivors (n = 589) Remaining in the Community or Institutionalized Long-term, 1975–84, According to Health Impairment and Disability in 1984

	Remaining in Community (n)	Institutionalized Long-term (n)	Percent of Impaired or Disabled Remaining in Community
Survivors	526	63	
Physical health			
4+ chronic conditions	124	16	88.6
<4 chronic conditions	402	47	
Cognitive impairment			
4+ errors	63	26	70.8*
<4 errors	443	10	
Psychological distress			
7+ symptoms	95	42	69.3*
<7 symptoms	431	21	
Functional disability			
dependent 1+ ADL	111	60	64.9*
not ADL dependent	415	3	

*p < .001 by chi square

TABLE 2—Logistic Regression of Demographic and Independent Variables, Measured in 1975, on Dependent Variable: Remaining in Community 1975–84, for Survivors (n = 589) and Deceased (n = 632)*

Variables	Odds Ratio	95% CL
Age (continuous, by year)	.93	.90–.96
Gender (M = 1)	2.70	1.72–4.16
Race (B = 1)	1.38	.87–2.17
Education (>8 yr. = 1)	.73	.50–1.07
Income ($5000+ = 1)	2.28	1.46–3.54
Lives with child(ren) only (= 1)	3.45	1.85–6.43
Physical health impairment (4+ chronic conditions = 1)	1.11	.68–1.82
Cognitive impairment (4+ errors = 1)	.85	.46–1.56
Psychological distress (>7 of 15 symptoms)	1.53	.87–2.70
Functional disability (help with 1+ ADL = 1)	.79	.52–1.20
Died between 1975 and 1984 (= 1)	.56	.38–.83

*82 cases omitted from analysis because of missing data.

tained for 589 of the living respondents (91 percent). By 1984, 780 individuals were known to have died. Telephone interviews with next of kin or other informants were obtained for 639 deceased, with data about institutionalization secured for 632 (81 percent).

Demographic information was collected according to the OARS instrument. Impaired physical health was indicated by the presence of four or more self-reported chronic conditions from a list of the 12 most prevalent conditions.[3] Impaired cognitive function was measured by a score of four or more errors on a 10-item test of cognitive function.[4] "Psychological distress" was measured as seven or more symptoms of a list of 15 on a Short Psychiatric Evaluation Schedule.[5] Functional disability was measured by need for help with one or more of six activities of daily living: walking, going outside, bathing, dressing, transfer (i.e. getting in or out of bed or chair), or eating.[6]

The definition of long-term institutionalization was one or more institutional admissions totaling 30 days or more during the nine-year observation period, or prior to death. The rest of the subjects were characterized as "remaining in the community," even though they may have experienced short-term admissions. The object was to distinguish those who continued to receive care mainly outside of institutions from those who entered long-term or indefinite institutional care.

Results

Comparison of the 1984 survivors with figures extrapolated from the 1980 census of Cleveland shows that the subjects, now aged 74 years and older, remained representative of the city population aged 75 and over.[7]

By 1984, 76 of the 647 responding survivors (11.8 percent) were living in an institution at the time of the interview, and a total of 63 (10.7 percent) of all respondents, including institutionalized and noninstitutionalized, had been admitted to a long-term care institution for 30 days or more. Of 731 persons who died during the nine-year observation period and for whom death certificates were available, 28.3 percent had a nursing home as their usual place of residence at the time of death.

Considering first the 589 survivors with complete data and their state of health impairment and functional disability at the time of reinterview in 1984, it appears that mental health impairment and functional disability were associated with institutionalization, but that the chances of remaining in the community were not affected by the presence of multiple chronic conditions. Expressed as a ratio of numbers of community residents to numbers of institutional residents, the figures are 7.8 for physical health impairment, 2.4 for cognitive impairment, 2.3 for psychological distress, and 1.8 for functional disability (Table 1).

The next stage of analysis was to ask whether it would have been possible to predict at the beginning of the study who would experience long-term institutionalization and who would remain in the community. A multivariate logistic regression was performed, using remaining in the community as the dependent variable, with independent variables as indicated in Table 2.

Factors significantly favoring continued care in the community include: younger age, male gender, income of greater than $5,000 (1975), and living with child(ren) only. Since previous analyses had pointed to living with others as favoring community care, three separate regressions were carried out, using different definitions of this variable. The odds ratio resulting were: lives with spouse, or children and spouse: 1.76 (95% CL = 1.08, 2.89), lives with others: 2.23 (95% CL = 1.54, 3.22), and lives with child(ren) only: 3.45 (95% CL = 1.85, 6.43).

Somewhat surprisingly, none of the four measures of impairment or disability served to distinguish between those who remained in the community and those who experienced long-term institutionalization. Those who died during the study period were less likely to have avoided long-term institutionalization.

Discussion

These results indicate that socioeconomic factors predominate as predictors of long-term care in the community. The way these factors operate is fairly clear. Relative youth implies better resources and coping skills. Males are more likely to have a surviving spouse. Better income allows for better housing and purchase of services, and children can be both care providers and income producers. In other recent longitudinal studies age, female gender, and living alone or absence of spouse have been shown to predict institutionalization.[8,9] Notably in the present study, when elders live with them, children appear to enhance the possibility of remaining in the community more than do living spouses or nonspecified "others."

Although, in this study, impairment and disability do not appear to predict whether community or institutional care will be used, it must be remembered that some part of nine years intervened between the measurement of dependent and independent variables. Observations closer to the decision-point might have shown that mental health impairment and functional disability were more difficult to care for at home than impairment of physical health alone, as suggested by the data in Table 1. ☐

Acknowledgments

Support for this project was provided by Grants P01 AG04391 and R01 AG07195 from the National Institute on Aging.

This report was presented in preliminary form at the World Congress of Gerontology, Acapulco, Mexico, June 22, 1989.

References

1. US General Accounting Office: The Well-Being of Older People in Cleveland, Ohio. HRD-77-70, Washington DC: US GAO, 1977.
2. Fillenbaum G: Multidimensional Functional Assessment of Older Adults: The Duke Older Americans Resources and Services Procedures. Hillsdale, NJ: Lawrence Erlbaum Associates, 1988.
3. Ford AB, Folmar SJ, Salmon RB, Medalie JH, Roy AW, Galazka SS: Health and function in the old and very old. J Am Geriatr Soc 1988; 36:187–197.
4. Pfeiffer E: A short portable mental status questionnaire for the assessment of organic brain deficit in elderly patients. J Am Geriatr Soc 1975; 23:433–441.
5. Kincannon JC: Prediction of the standard MMPI scale scores from 71 items: The mini-mult. J Consult Clin Psychol 1968; 32:319–325. (Modified by Pfeiffer—see reference 2.)
6. Feller BA: Americans Needing Help to Function at Home. National Center for Health Statistics, Advance Data from Vital and Health Statistics, No. 92, Department of Health and Human Services Publ. No. (PHS) 83-1250. Hyattsville, MD: National Center for Health Statistics, 1983.
7. Ford AB, Haug MR, Jones PK, Roy AW, Folmar SJ: Race-related differences among elderly urban residents: A cohort study, 1975–1984. J Gerontol Soc Sci 1990; 45: 5163–5171.
8. Branch LG, Jette AM: A prospective study of long-term care institutionalization among the aged. Am J Public Health 1982; 72:1373–1378.
9. Shapiro E, Tate RB: Predictors of long-term care facility use among the elderly. Can J Aging 1985; 4:11–19.

Recent Trends in the Incidence of Toxic Shock Syndrome in Northern California

Diana B. Petitti, MD, MPH, and Arthur L. Reingold, MD

ABSTRACT

The incidence of toxic shock syndrome in women members of a large prepaid medical care program in Northern California was 1.5 cases per 100,000 in a period after removal of tampons containing polyacrylate rayon and reductions in tampon absorbency. This rate was lower, but not significantly lower, than the rate of 2.2 per 100,000 in the prior interval. It was higher, but not significantly higher, than the rate of 0.4 per 100,000 in the era before "superabsorbent" materials were introduced into tampons. The incidence in men has been stable at about 0.1 cases per 100,000 for the 15-year period from 1972 though 1987. (*Am J Public Health.* 1991;81:1209–1211)

Introduction

We previously reported the incidence of toxic shock syndrome (TSS) through 1985 in members of the Northern California Kaiser Permanente Medical Care Program ages 15–34 years.[1–3] Here, we present a final update on TSS in this population, presenting data for 1986 and 1987. The incidence of TSS in these latter two years is of particular interest, because polyacrylate rayon, a material included in tampons to increase their absorbency, was removed from tampons marketed in the United States in March 1985 and because there were further declines in the absorbency of tampons overall. Since high absorbency has been linked with an increase risk of TSS in tampon users,[4] one would expect the incidence of TSS to decline in women after reductions in the absorbency of tampons brought about, at least in part, by removal of polyacrylate rayon from tampons.

Methods

Our method of case ascertainment, in which medical records were reviewed to identify recognized and unrecognized cases of TSS, was described in detail in a

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TABLE 1—Incidence of Definite TSS in Females Ages 15–34 in Relation to Certain Milestones in Tampon Formulation and Marketing

Time Periods	Description	Number of Cases	Incidence* (95% Confidence Interval)
1. 1972–June 1977	Prior to introduction of superabsorbent materials	5	0.4 (0.1,1.0)
2. July 1977–April 1979	After superabsorbents introduced, but before Rely marketing in California	8	1.5 (0.6,2.9)
3. May 1979–September 1980	Rely and other superabsorbents in California	11	2.4 (1.2,4.2)
4. October 1980–March 1985	After Rely removal from the market but before removal of polyacrylate rayon	34	2.2 (1.5,3.0)
5. April 1985–December 1987	After removal of polyacrylate rayon products and reductions in absorbency	14	1.5 (0.8,2.6)

* per 100,000 women-years

prior publication.[1] Briefly, we used computer-stored information from records of discharges from Northern California Kaiser Permanente Medical Care Program facilities for the period 1972 through 1987 to identify men and women ages 15–34 years hospitalized with illnesses that might be confused with TSS, or, for later years, with diagnosed TSS. Records for qualifying hospitalizations were reviewed by a single trained medical record technician, who determined whether or not the illness met the case definition for TSS developed by the Centers for Disease Control (CDC), except that desquamation and orthostatic dizziness were not considered criteria because desquamation was not reliably recorded in the medical records and because orthostatic hypotension was not an official criterion for TSS when the study began. Records for all illnesses that might represent TSS were then reviewed independently by each of the co-authors, who classified them as definite TSS, probable TSS, or not TSS. Definite TSS cases were illnesses that met all the CDC criteria as TSS, except desquamation. Probable cases were illnesses that had rash, fever, and hypotension, but involved only two organ systems.

The denominators for the calculation of the incidence rates were person-years of membership in the Northern California Kaiser-Permanente Medical Care Program. Ninety-five percent confidence intervals were estimated based on the assumption that the incidence of TSS is distributed as a Poisson. Statistical tests of the hypotheses about rates of TSS were carried out based on the comparison of two Poisson variables.

Results

Table 1 shows the incidence of definite TSS in women in relation to certain milestones in tampon formation and marketing. The incidence of TSS was statistically significantly higher (p = 0.0003) after introduction of superabsorbent materials and Rely tampons into the California market (period 3) than in the period prior to these two changes in tampon for-

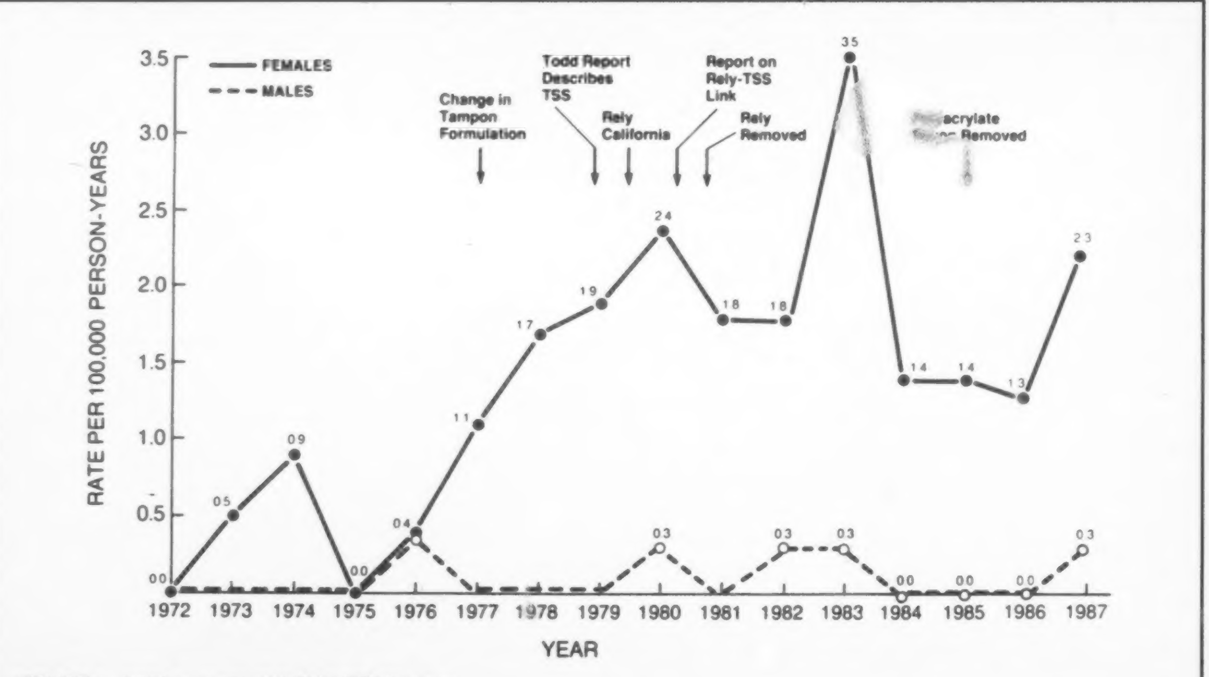

FIGURE 1—Incidence rate of definite TSS by year.

mulation and marketing (period 1). The incidence of definite TSS in women after removal of Rely and polyacrylate rayon from remaining tampons (period 5) was lower, but not significantly lower ($p = 0.13$) than in the prior two intervals (periods 3 and 4). The incidence rate after removal of polyacrylate (period 5) was also not statistically significantly higher ($p = 0.13$) than in the period prior to introduction of superabsorbent materials (period 1).

Information on menstrual status at the onset of symptoms was retrievable from the medical records of all 12 definite TSS cases in women in 1986 and 1987 (Figure 1). Eight of these cases (67 percent) were menstrual (95% CI = 49, 85). This percentage is the same as it was in prior years in this population.[2]

The incidence of TSS in men has been stable at about 0.1 cases per 100,000 throughout the 15-year period covered by this surveillance project.

Discussion

The incidence of TSS began increasing in about 1977 in temporal relation to changes in tampon formulation that made them more absorbent. The rarity of TSS in this population limits the ability to draw firm conclusions about the incidence of TSS in relation to the removal of Rely tampons from the market, the subsequent removal of polyacrylate rayon, and the reductions in the absorbency of tampons that occurred simultaneously. The data are suggestive of a decline in incidence in relation to removal of polyacrylate rayon and accompanying reductions in absorbency. However, these changes in tampon formulation have clearly not eliminated TSS in women.

It is reasonable to conclude TSS is very rare in men. It also appears that in this population the percentage of menstrual cases is stable at about two-thirds of all cases in women. The incidence in both men and women in this population is very low, and TSS is not an important public health problem at this time. ☐

Acknowledgments

This research was supported by a grant from the California Public Health Foundation with funds from the Proctor & Gamble Corporation. We are indebted to Beverly Whitmarch for reviewing the medical records.

References

1. Petitti DB, Reingold A, Chin J: The incidence of toxic shock syndrome in Northern California, 1972 through 1983. JAMA 1986; 255:368–372.
2. Petitti DB, Reingold A: Tampon characteristics and menstrual toxic shock syndrome. JAMA 1988; 259:686–687.
3. Petitti DB, Reingold A: Update through 1985 on the incidence of toxic shock syndrome in members of a prepaid health plan. Rev Infect Dis 1989; 11(Suppl):22–26.
4. Berkley, SF, Hightower AW, Broome CV, Reingold AL: The relationship of tampon characteristics to menstrual toxic shock syndrome. JAMA 1987; 258:917–920.

Hospital Charges in Five Washington Counties Jump Average of Nearly 37%

Hospital charges in the state of Washington's five largest counties increased by an average of nearly 37% between 1987 and 1989, according to a report by the Washington State Department of Health.

The report, "Inflation Measures in Hospital Charges," covers 15 separate categories, from coronary bypass surgery to Caesarean delivery, at hospitals in King, Pierce, Snohomish, Thurston, and Spokane Counties.

"Hospital charges climbed at a significant rate over that 3-year period," said State Health Secretary Kristine M. Gebbie. "This report confirms that the issues surrounding the inflation of hospital charges are complex and that the upper range of these inflation measures are well above the consumer price index or even average increases in medical charges."

Increases in charges over the 3-year period range from 9.9% for treating alcohol and drug dependency to 67.7% for newborn children with major medical problems. Annual inflation figures for those same categories of treatment were 4.1% and 32.8%, respectively, with a median annual inflation rate of 10.5% covering all 15 categories.

The overall average increase for the 15 categories of services was 36.6% over the 3-year period.

The amount of inflation varied by category of service and the county. For example, average charges for cardiac bypass surgery in King County jumped from about $17,500 in 1987 to just over $22,200 in 1989, whereas in Pierce County the charges for similar procedures increased from just under $23,000 to more than $30,000. In Spokane County, similar bypass surgery jumped from about $18,800 to about $21,500.

For this type of bypass surgery, the overall average charge increased from $19,038 to $23,482, or about 23.3%. Annually, this meant the inflation rate for this procedure was 10.7%. The greatest total increase over the 3-year period was 30.8% in Pierce County, and the smallest gain was in Spokane County with 14.6% over 3 years.

Another category, care for newborn children without significant problems, represents the greatest single area of competition between hospitals, according to the state agency's report.

The average charge for newborn care within the five counties increased from $478 for the first half of 1987 to $555 for the second half of 1989, or about 16%. This level of increase did not occur in all counties. Snohomish and Spokane counties posted 20.8% and 28.5%, respectively.

Two measures of inflation are used extensively throughout the report: total change in the average charge from the first half of 1987 through the last half of 1989; and the average annual rate for 1988 and 1989. The report also measures the average level of inflation in charges covering all patients and all categories.

In addition to bypass surgery and Caesarean section deliveries, the 15 categories of service include chemotherapy, reattaching major joints or limbs, and psychiatric care, including the treatment of schizophrenia and manic depression.

Anyone who wants a copy of the report may contact the State Department of Health, Office of Hospital Data Systems, Mail Stop FJ-21, Olympia, WA, 98504, or phone 206/753-1990 during regular business hours.

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American Journal of Public Health

American Public Health Association

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encompassing approach, a renewal of enthusiasm for control of the "other STDs." ☐

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References

1. Cates W. The "Other STDs": do they really matter? *JAMA*. 1988;259:3606–3608.
2. Cates W. Epidemiology and control of sexually transmitted diseases. In: Handsfield HH, ed. *Infectious Disease Clinics of North America, Vol 1*. Philadelphia, PA: WB Saunders Co; March 1987:1–24.
3. Rice RJ, Roberts PL, Handsfield HH, Holmes KK. Sociodemographic distribution of gonorrhea incidence: implications for prevention and behavioral research. *Am J Public Health*. 1991;81:1252–1258.
4. Yorke JA, Hethcote HW, Nold A. Dynamics and control of the transmission of gonorrhea. *Sex Transm Dis*. 1978;5:51–56.
5. Hethcote HW, Yorke JA. Gonorrhea transmission dynamics and control. *Lecture Notes in Biomathematics No. 56*. Berlin: Springer-Verlag; 1984.
6. Rothenberg RB. The geography of gonorrhea: empiricial demonstration of core group transmission. *Am J Epidemiol*. 1983; 117:688–694.
7. Rothenberg RB, Potterat JJ. Temporal and social aspects of gonorrhea transmission: the force of infectivity. *Sex Transm Dis*. 1988;15:88–92.
8. Zenilman JM, Bonner M, Sharp KL, Rabb JA, Alexander ER. Penicillinase-producing Neisseria gonorrhoeae in Dade County, Florida. Evidence of core group transmitters and the impact of illicit antibiotics. *Sex Transm Dis*. 1988;15:45–50.
9. Arya OP, Rees E, Turner CG, et al. Epidemiology of penicillinase-producing Neisseria gonorrhoeae in Liverpool from 1977 to 1982. *J Infect Dis*. 1984;8:70–83.
10. US Department of Health and Human Services. *Promoting Health/Preventing Disease*. Washington, DC: US Government Printing Office; 1980.
11. Rothenberg RB. Epidemiologic aspects of control of penicillinase-producing Neisseris gonorrhoeae. *Sex Transm Dis*. 1988;15:211–216.
12. Schachter J. Chlamydia infections. *New Engl J Med*. 1978;298:428–435, 490–495, 540–549.
13. Thompson SE, Washington AE. Epidemiology of sexually transmitted Chlamydia trachomatis infections. *Epidemiol Rev*. 1983;5:96–123.
14. Stamm WE. Diagnosis of Chlamydia trachomatis genitourinary infections. *Ann Int Med*. 1988;108:710–717.
15. Centers for Disease Control. *Sexually Transmitted Disease Surveillance, 1989*. Atlanta, Ga: Centers for Disease Control; 1990.
16. Rolfs RT, Goldberg M, Sharrar RB. Risk factors for syphilis: cocaine use and prostitution. *Am J Public Health*. 1990;80:853–857.
17. Lukehart SA, Hook EW III, Baker-Zander SA, Collier AC, Critchlow CW, Handsfield HH. Invasion of the central nervous system by Treponema pallidum: implications for diagnosis and therapy. *Ann Intern Med*. 1988;109:855–862.
18. Gissman L, Durst M, Oltersdorf, von Knebel Doeberitz M. Human papillomaviruses and cervical cancer. In: Steinberg BM, Brandsma JL, Taichman LB, eds. *Cancer Cells/5 Papillomaviruses*. Cold Spring Harbor, Mass: Cold Spring Harbor Laboratory; 1987:275–280.
19. Centers for Disease Control. Program Announcement No. 121: Early detection and control of breast and cervical cancer. *Federal Register*. May 8, 1991;56(89):21379–21383.

Erratum

In: Bartlett AV, Englender SJ, Jarvis BA, Ludwig L, Carlson JF, Topping JP. Controlled trial of *giardia lamblia:* control strategies in day care centers. *Am J Public Health*. 1991;81:1001–1006.

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Sociodemographic Distribution of Gonorrhea Incidence: Implications for Prevention and Behavioral Research

Roselyn J. Rice, MD, Pacita L. Roberts, MS, H. Hunter Handsfield, MD, and King K. Holmes, MD, PhD

ABSTRACT

Background. Despite a declining incidence during the AIDS era, gonorrhea remains the most frequently reported communicable disease in the United States.

Methods. During 1986 and 1987 we supplemented gonorrhea case reporting with laboratory surveillance in King County, Washington. Incidence rates were correlated with demographic variables.

Results. Overall incidence of gonorrhea was similar for men and women, but highest for 16- to 21-year-old females and urban Seattle residents. Incidence rates by ethnicity were Blacks, 3033; Native Americans, 843; Hispanics, 617; Asians, 190; and Whites, 121. Census tracts representing the lowest socioeconomic status (SES) quartile accounted for 58% of reported gonorrhea. Black female teenagers residing in the lowest SES urban areas had highest incidence rates: aged 14 to 15, 3.4%; 16 to 17, 10.4%; 18, 17.0%; and 19, 15.4%. Rates in female teenagers were even higher after adjustment for estimated proportion of those who were sexually experienced.

Conclusions. Gonorrhea incidence is associated with age, gender, ethnicity, SES, and residence. Identification of populations at highest risk for gonorrhea can direct interventions against all sexually transmitted diseases. Clearly, interventions to alter high-risk behaviors must be initiated in early adolescence. (*Am J Public Health*. 1991;81:1252–1258)

Introduction

With the advent of effective contraception, the onset of sexual maturity in the baby boom generation, and changes in sexual behavior, reported cases of gonorrhea increased at about 15% per year from the 1960s through the mid-1970s in the United States.[1,2] Reported cases have gradually fallen since 1975.[3] After 1982 a steady decline in rectal gonorrhea in men was observed in King County, Washington, and in other areas of the United States,[4,5] implying a decline in gonorrhea among homosexually active men. Further analysis of trends in occurrence of gonorrhea by age, gender, and race for the period 1980 through 1988 in King County has shown more than a threefold decline in cases occurring in White men, little change in White women, but a steady increase in Black men and women from 1982 through 1988 (authors' unpublished data). Among Blacks, the greatest increase in annual cases has occurred among teenagers. This unexpected increase in gonorrhea in Blacks and the concurrent leveling off of reported cases in White women after nearly a decade of decline led us to undertake a 2-year intensive surveillance of gonorrhea in Seattle-King County to define incidence rates in population subgroups according to age, gender, race or ethnicity, residence (urban, suburban, or rural), and socioeconomic status (SES). The results are useful not only for defining more precisely a current profile of individuals at highest risk for gonorrhea, but also as a proxy measure of the distribution of sexual behaviors that place individuals at risk for all sexually transmitted diseases, including the acquired immunodeficiency syndrome (AIDS).

Methods

Surveillance and Case Reporting

King County, Washington (population 1.4 million, 2130 square miles), encompasses the city of Seattle (population 496 000). Health care providers in King County are required to report all gonorrhea cases; compliance is 100% by Seattle-King County Department of Public Health (DPH) clinics, but variable by other clinics and the private health care sector.

Surveillance

Beginning in 1985, presentations to microbiology laboratory directors, monthly bulletins of the DPH, newsletters and bulletins of the King County Medical Society, and hospital staff meetings were used to publicize the enhanced gonorrhea surveillance project and encourage compliance with effective culture diagnosis and case reporting. Routine case reporting was supplemented by active laboratory surveillance for *Neisseria gonorrhoeae* in

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This paper was submitted to the journal March 21, 1990, and accepted with revisions February 26, 1991.

Editor's Note. See related Editorial by Richard Rothenberg on page 1250 of this issue of the journal.

laboratories in King County through a system established with the DPH central laboratory. Hospital, private physician, and commercial laboratories were asked to submit all positive and confirmatory culture isolates for *N. gonorrhoeae* to the DPH laboratory for confirmation and antimicrobial susceptibility screening tests.

The DPH processed 56 353 cultures for *N. gonorrhoeae* in 1986 and 52 844 in 1987. During 1986 and 1987, gonococcal isolates were received from 14 hospitals, 4 private microbiology laboratories, 2 student health clinics, 2 military health clinics, all 12 DPH clinics, 22 community clinics (partially supported by DPH), the Seattle-King County Jail and Juvenile Detention Center, 6 private family planning clinics, and numerous physicians' offices. Of the cultures confirmed as positive for *N. gonorrhoeae*, 47.0% were submitted from DPH clinics, 22.2% from private physicians, 20.4% from hospitals, 6.0% from community clinics, 3.6% from private family planning clinics, 0.6% from student health clinics, and 0.1% from military clinics. Matching of positive cultures confirmed by the DPH laboratory with submitted case reports from all public and private providers indicated that cultures were submitted to the DPH laboratory for confirmation from 78% of the reported gonorrhea cases in the study population.

Case Reporting

Gonorrhea case reports were submitted to the DPH using Centers for Disease Control (CDC) form 73.688 (revised 1/86). Following notification of a case of gonorrhea by case report form or as a result of laboratory surveillance, when necessary, DPH staff contacted health care providers for additional demographic information on age, gender, race or ethnicity, address, anatomic site of infection, date of infection, date of treatment, and clinical diagnosis (asymptomatic gonorrhea, symptomatic gonorrhea, pelvic inflammatory disease [PID], disseminated gonococcal infection [DGI], adult conjunctivitis).

Demographic Classification

Data from case reports were abstracted and analyzed for all individuals with gonorrhea who resided in King County at the time of diagnosis. Residence was classified as urban for persons residing within the geographic boundaries of Seattle and as suburban for persons living in incorporated areas of King County or in census tracts with population densities of more than 2000 persons per square mile (based on 1980 US Census statistics). The remainder were classified as rural King County residents. Race and ethnicity were classified according to the CDC case report form 73.688 as White, Black, Hispanic, Asian/Pacific Islander, Native American/Eskimo, or other. Hispanics were classified separately, regardless of race designation.

Socioeconomic Categorization of Census Tract of Residence

Census tracts in Seattle were divided into four socioeconomic categories on the basis of the following population characteristics summarized from the 1980 US Census on Population and Housing[6]: (1) median family income; (2) percent that completed high school; (3) percent employed in professional, managerial, or technical occupations; (4) housing cost index; and (5) percent in owner-occupied housing. A score was computed for each tract from the sum of the tract rankings according to the stated characteristics, and these scores were used to categorize tracts into low, lower-middle, upper-middle, and high SES groupings. These four groupings comprised 25.5%, 28.2%, 18.7%, and 27.6% of the city population, respectively.

Calculation of Gonorrhea Incidence

Incidence rates for gonorrhea were calculated using the total number of cases reported to the Seattle-King County DPH in the 2-year period 1986 to 1987, divided by the total county population count from the 1980 US Census of King County population. This was halved to arrive at an annual rate. Incidence rates by gender, age, race or ethnicity, and census tract of residence were calculated in the same way, using 1980 US Census Bureau data for King County.[6]

Calculation of Estimated Incidence Rates by Marital Status

Marital status was not known for 50% of cases reported from sources other than DPH clinics. In order to estimate incidence rates for marital status, the distribution of marital status for cases where this was known was applied to the unknowns.

Calculation of Adjusted Incidence Rates for Sexually Experienced Women

Gonorrhea rates were calculated for women of White and other races using as denominators national estimates of the number of women who were sexually experienced, as reported in the 1982 National Survey of Family Growth Report;[7] these estimates were adjusted for the number of women in King County in 1980 by age and race.

Statistical Analysis

Correlation between incidence rates and population density in census tracts was examined statistically by computing the correlation coefficient (r).

Results

Incidence of Gonorrhea by Age, Gender, Race, Residence, and Marital Status

Gender and Age Group. The overall annualized incidence rates of gonorrhea by gender were 293 per 100 000 for men and 286 per 100 000 for women. As summarized in Figure 1, the incidence of gonorrhea was highest in females aged 16 to 19 years. For ages 16 to 17 years, incidence rates of gonorrhea were four times higher for females than for males. In contrast, gonorrhea incidence among men was more broadly distributed across age groups, but was higher for men than for women above age 24. Men and women in the 22- to 24-year-old age group had similar rates of gonorrhea.

Race and Ethnicity. Of the 7342 reported gonorrhea cases, 39.6% involved Whites, 48.8% Blacks, 3.3% Native Americans, 3.5% Asians and Pacific Islanders, and 4.9% Hispanics. The annual incidence of gonorrhea per 100 000 by race or ethnicity is shown in Table 1.

There were remarkable differences among racial and ethnic groups in the age and gender distribution of gonorrhea incidence. Among men, the peak incidence of gonorrhea per 100 000 was observed at age 19 for Blacks (11 266) and Native Americans (2299), age 20 for Asians and Pacific Islanders (807), age 21 for Whites (283), and ages 22 to 24 for Hispanics (2597). Among women, the peak incidence per 100 000 was observed at age 18 for Blacks (12 848), Whites (729), and Hispanics (906), and age 19 for Native Americans and Eskimos (6250) and Asians and Pacific Islanders (1068). The overall male:female ratio for gonorrhea incidence was 1.18 for Blacks, 0.73 for Whites, 0.48 for Native Americans, 1.50 for Asians and Pacific Islanders, and 3.51 for Hispanics. As shown in Figure 2, the gender ratio for gonorrhea incidence followed a similar pattern for Whites and Blacks, with females predominating at younger ages and males predominating at ages 25 to 29 years

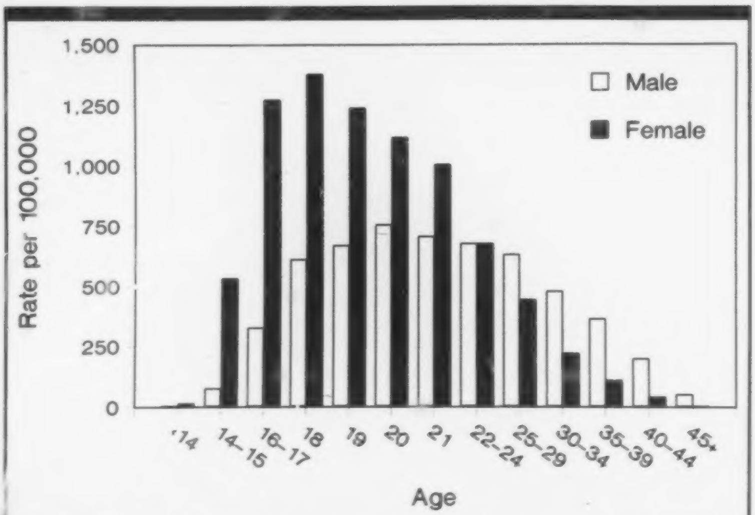

FIGURE 1—Annual incidence of gonorrhea per 100 000 population for Seattle-King County residents by gender and age group, 1986–1987.

TABLE 1—Annual Incidence of Gonorrhea per 100 000 Urban Residents of Seattle According to Race or Ethnicity and Socioeconomic Status (SES), 1986 and 1987

Race or Ethnic Group	SES of Residence Census Tract				All SES Groups
	Low	Lower-Middle	Upper-Middle	Upper	
White	386	153	104	78	121
Asian	310	214	181	125	190
Hispanic	1796	527	427	302	617
NA[a]	1961	1003	640	519	843
Black	3394	3018	2305	1990	3033
All race or ethnic groups	1147	421	224	183	289

[a]NA=Native American.

and above for both groups. However, for Hispanics, rates for females exceeded rates for males only for ages 17 years and under and were about threefold to sixfold lower after age 21. Similarly, for Asians and Pacific Islanders, the incidence in males exceeded that for females for all ages after age 19 and remained low thereafter. In contrast, for Native Americans the incidence rates for females were higher than incidence rates for males for all ages up to 35 years. These differences are large, but are based on relatively small numbers of cases involving Hispanics, Asians and Pacific Islanders, and Native Americans and Eskimos. The differences could also be influenced by underreporting of male cases, as discussed below.

Marital Status. The annual adjusted incidence of gonorrhea per 100 000 was 42 for married men; 67 for divorced, separated, or widowed men; and 1018 for never-married men. The incidence per 100 000 was 33 for married women, 21 for divorced, separated, or widowed women; and 1275 for never-married women. We did not adjust further for marital status in subsequent analyses because data on marital status were not available for all cases.

Urban, Suburban, and Rural Residents. Of the 7342 gonorrhea cases included in this analysis, 74% occurred in urban (i.e., Seattle) residents, 19% in residents of suburban census tracts with ≥2000 residents per square mile, and 7% in residents of rural census tracts (<2,000 residents per square mile). The annualized incidence rates per 100 000 were 555 for urban residents, 143 for suburban residents, and 86 for rural residents.

Socioeconomic Status. In Seattle, which we defined as urban, the population living in low SES census tracts represented only 25.5% of the population but accounted for 58% of the reported gonorrhea. There were 2870 cases in 125 143 residents of low SES census tracts, 1164 cases among 138 294 residents of lower-middle SES census tracts, 409 cases among 91 407 residents of upper-middle SES census tracts, and 494 cases among 135 262 residents of high SES census tracts.

Annual incidence rates according to race or ethnicity and SES are shown in Table 1. Within each racial or ethnic group, there was an inverse and statistically significant relationship between gonorrhea incidence and the SES of the census tract of residents ($P<.01$ for each group). Within each SES grouping, the relative incidence rates by race and ethnicity generally corresponded to the unadjusted relative rates, but the magnitude of the differences by race and ethnicity was reduced. For example, the overall incidence rate was 25 times greater for Blacks than for Whites, but among those of lowest SES the rate was 8.8 times greater for Blacks than for Whites.

Incidence in Female Residents of Low SES Urban Census Tracts. Because the highest incidence of reported gonorrhea occurred in urban census tracts of low SES, and because the completeness of surveillance for laboratory-confirmed gonorrhea may have been highest for females (see Discussion), we further analyzed the incidence rates among women within these census tracts according to age and race or ethnicity. As shown in Figure 3, the highest incidence rates were observed in Black female teenagers, among whom the highest annual incidence reached 17 040 per 100 000 at age 18 and declined thereafter.

Correlation of Gonorrhea Distribution and Incidence with Population Density and Residence. The incidence of gonorrhea by census tract was directly but weakly correlated with population density ($r=.55$, $P=.02$). Mapping of gonorrhea incidence indicated two clusters of high-incidence census tracts, each with ≥1000 cases of gonorrhea per 100 000 population per year, in central and south Seattle (Figure 4). These clusters corresponded to those tracts with the lowest SES rankings and with the highest proportions of non-White residents.

Estimated Incidence Rates in Sexually Experienced Women. By adjusting the denominators to represent only the estimated number of women who were sexually experienced,[7] as advocated by others,[8] the greatest effect on estimated incidence rates was observed, as expected, among teenagers. Regardless of race, adjusted incidence rates increased by about four- to sixfold in 15-year-old adolescent women and by 1.2 to 2.9 in groups of 16- to 17-, 18-, and 19-year-old women (Table 2).

Distribution of Cases by Provider Source. The distribution of gonorrhea by gender, age, and race or ethnicity was compared for cases reported from (1) DPH clinics; (2) hospitals, community clinics, and private physicians; and (3) private family planning clinics. Public clinics reported 56% of cases in males and 38% of cases in females. DPH clinics reported 991 (68%) of 1457 cases in Black men aged over 20 years, vs 545 (58%) of 942 cases in White men of the same age. Otherwise, the distribution of cases by age, sex, and race was not substantially different between DPH clinics and the other groups of providers.

Complications of Gonococcal Infection. Of all women with gonorrhea, 18% received a clinical diagnosis of PID, including 20% of Native Americans, 19% of Whites, 18% of Blacks, and 8% of Hispanics. Fifteen cases of DGI were reported, representing 0.2% of all gonorrhea cases; women accounted for 12 (80%) of these 15 cases. Six cases of adult gonococcal conjunctivitis were diagnosed during the 2-year period. Data were inadequate for estimating the incidence of epididymitis.

Discussion

The distribution of gonorrhea incidence in King County varies markedly according to age, gender, race or ethnicity, and residence. Among individuals classified by age and gender, by far the highest incidence was observed for 18-year-old women, and among adolescents, the ratio of female to male cases was highest for those 14 to 17 years of age. This fact would be obscured by aggregating data into 5-year age groups as in national data sets for sexually transmitted diseases. In explaining these differences in gonorrhea incidence in adolescent males and females, the relative contribution of differences in exposures and differences in host susceptibility remains to be determined. For men and women, the incidence of gonorrhea is highest among certain ethnic minorities living in urban, lower SES census tracts.

A critical feature of this analysis is the adequacy of surveillance. Was case detection representative and complete, or was it incomplete and biased? Evidence for completeness in surveillance includes

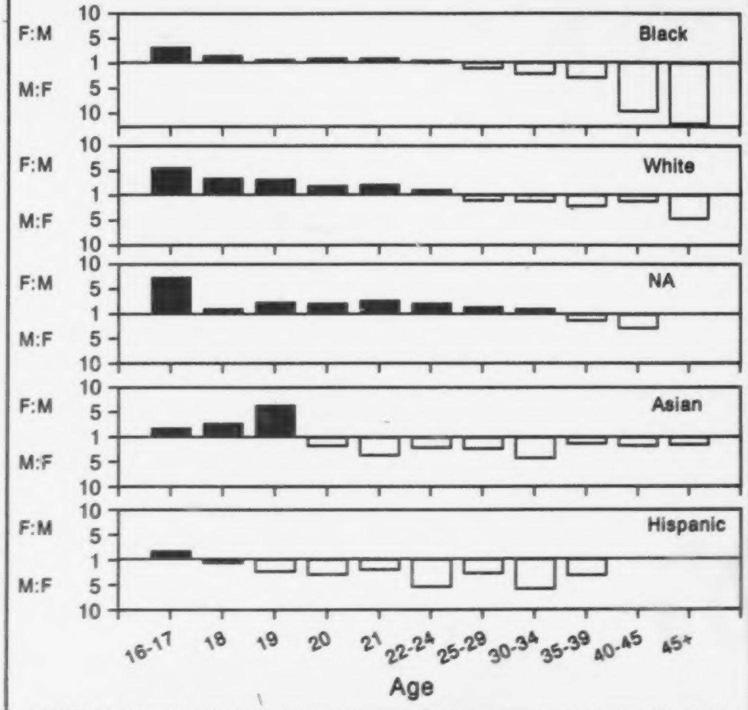

FIGURE 2—Gender ratio for incidence of gonorrhea by age and race or ethnicity. Solid bars above the line indicate a higher incidence in females than in males for that age and race or ethnic group; open bars below the line indicate a higher incidence in males than in females. NA = Native American Indian.

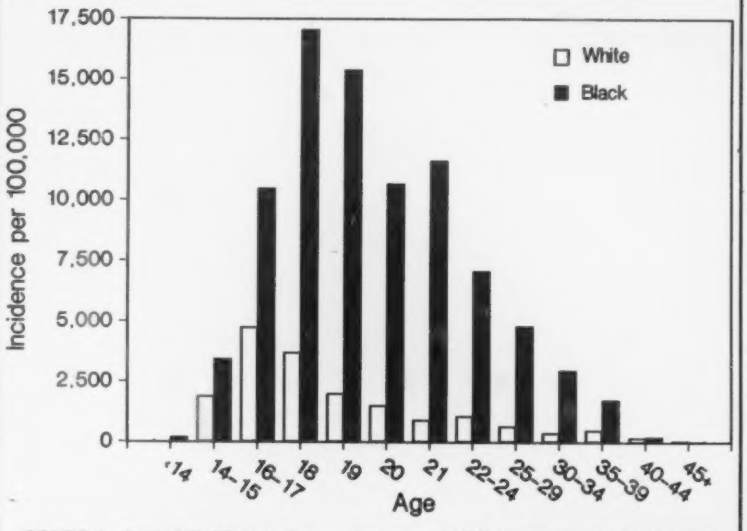

FIGURE 3—Annual incidence of gonorrhea per 100 000 female residents of lower socioeconomic status census tracts in urban Seattle, by age and race (White vs Black), 1986–1987.

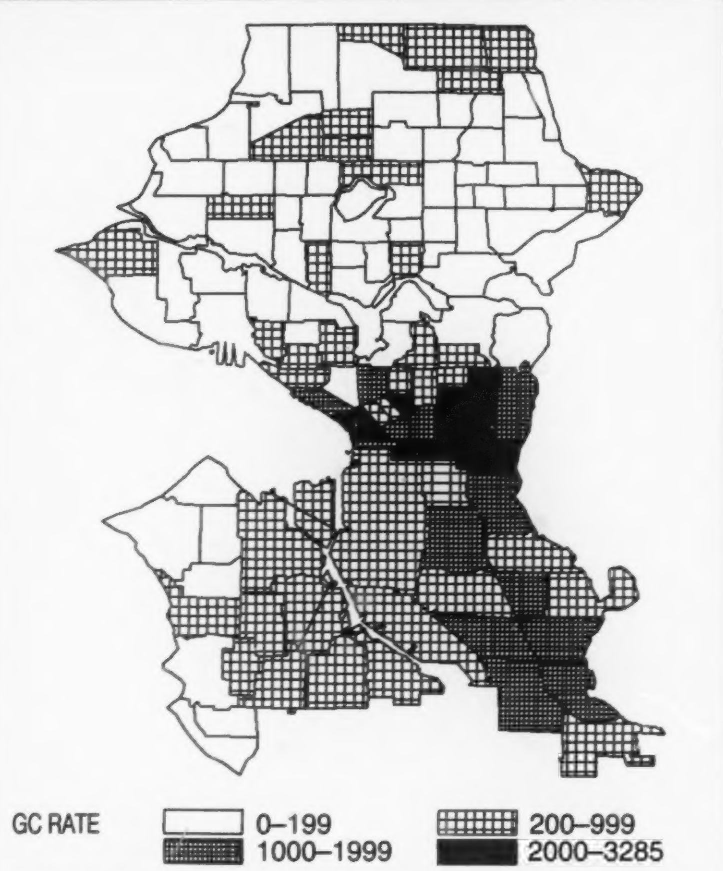

FIGURE 4—Map illustrating the geographic distribution of gonorrhea incidence rates (GC Rate) by census tract of residence in urban Seattle, 1986–1987.

TABLE 2—Gonorrhea Incidence Rates per 100 000 for White and Black Women in King County by Age, Unadjusted and Adjusted for Sexual Experience[a]

	White			Black		
Age	Unadjusted Incidence	% Sexually Experienced	Adjusted Incidence	Unadjusted Incidence	% Sexually Experienced	Adjusted Incidence
14	244	—	—	2395	—	—
15	367	17.9	2051	3873	28.0	13833
16–17	630	35.1	1796	11810	50.0	23621
18	729	54.8	1331	12849	77.0	16686
19	696	69.0	1000	11173	82.0	13625
20	528	84.5	624	10320	93.2	11073
21	590	84.5	698	8687	93.2	9321
22–24	357	84.5	423	6400	93.2	6867

[a] Crude and adjusted gonorrhea incidence rates did not change substantially among women aged ≥ 25 years.[9]

the one-to-one male to female ratio of reported cases; the similar distribution by age, gender, and race or ethnicity of cases from various reporting sources, with the exception noted; and the fact that 53% of cases (44% of males and 62% of females) were reported from non-DPH sources. Nationally, for 1987, only 32% of cases of gonorrhea were reported by private providers.[3] Further, virtually all community clinics, which are partially supported by DPH, use the DPH laboratory for gonorrhea culture diagnosis. Almost all major hospitals and their associated clinics in King County reported cases of gonorrhea through their laboratories during the study period, and these laboratories as well as most commercial laboratories submitted gonococci to the health department laboratory for confirmation and determination of antimicrobial susceptibility.

Regarding reporting of gonorrhea in males, in a unique study of gonococcal urethritis in King County that involved active surveillance of male patients of a sample of private physicians during 1974 and 1975, it was estimated that 38% of cases of gonococcal urethritis were seen in DPH clinics and 62% by all other reporting sources.[9] Some cases in men seen by non-DPH providers were identified by Gram-stained smear of urethral exudate. The proportion of cases of gonorrhea in men that was seen by non-DPH providers in the present study (44%) was lower than the 1974–1975 estimate, and we assume that some cases of gonorrhea in men are still diagnosed by Gram stain, so that no gonococcal isolate would have been sent to the DPH laboratory. It is thus probable that the proportion of cases of unreported gonorrhea seen in the private sector is somewhat higher for men than for women. Thus, we are most confident of the surveillance data for women. It is likely that some of the differences between racial and ethnic groups in the female-to-male ratio for gonorrhea incidence rates could be influenced by underreporting of gonorrhea in men seen in the private sector. However, we think it is unlikely that this could account for the major differences between racial minority groups in the female-to-male ratio and should not account for the steady decline in the female-to-male ratio with increasing age that was observed for most groups.

Even with enhanced surveillance of gonorrhea, cases in males most likely represent incident cases (new infections), while cases in females represent a mixture of incident cases and prevalent cases of uncertain duration: the shorter the duration, the closer the approximation to incident cases. Thus the comparison of male rates to female rates is not straightforward. It is uncertain how age and race or ethnicity influence the duration of infection at the time of diagnosis among women, but again, it seems unlikely that such influences would account for the differences shown in Figure 2.

The 1980 census data for King County were used to estimate the size of the population subgroups at risk during 1986 and 1987. From 1980 to 1986, it is estimated that the total population of King County increased by 7.2%, but that of Seattle increased by only 0.4%. It is possible that unequal trends in the size of the King County population belonging to individual racial and racial or ethnic groups between 1980 and the 1986-1987 period of study could lead to overestimates or underestimates of the calculated incidence of gonorrhea. The Office of Financial Management Forecasting Division, State of Washington,[10] in fact, provides estimates on changes in the size of the population of King County by race and Hispanic origin. These estimates are based on the "component method," which adds births, subtracts deaths, and adds the estimated net migration as estimated from school enrollments.

Because information on Hispanic origin, which is not a category of race, was not collected on vital records or school records, estimates of Hispanic population were derived by taking the 1980 census proportions of persons of Spanish origin in individual racial groups and applying them to the 1986 population estimates for racial groups. Washington State estimates that in King County from 1980 to 1986 the number of Blacks increased by 12%; Whites increased by 5%; Hispanics by 36%; Asians and Pacific Islanders by 26%; and Native Americans by 11%. Thus, the calculated incidence figures by race and Hispanic origin as presented in Table 2 probably represent substantial overestimates for Hispanics and Asians/Pacific Islanders, less substantial overestimates for Blacks, and small overestimates for Native Americans and Whites. Nonetheless, the relative incidence of gonorrhea in these racial and ethnic groups remains essentially as indicated in Table 1.

National data on gonorrhea incidence have not been published by race and ethnicity in recent years. Some states do not report data on race and ethnicity to the CDC for data aggregation and analysis. Nonetheless, in an analysis of gonorrhea incidence in New York State, Rothenberg[11] observed that "core" census tracts accounted for a disproportionately high percentage of gonorrhea cases. These were predominantly inner-city, low-SES census tracts with high density populations of ethnic minorities. It has been theorized that such core group populations are important in maintaining a high endemic level of gonorrhea, and that gonorrhea transmission is strongly affected by behavioral interactions within these population subgroups.[12,13] We observed a very strong inverse correlation of gonorrhea incidence with the SES rank of census tract of residence, despite nonhomogeneity with respect to various measures of SES of individuals within census tracts; this was independent of race and ethnicity. Although large differences in incidence rates between racial or ethnic groups persisted after adjustment for SES, the nonhomogeneity of SES for individuals within census tracts represents a limitation of these analyses.[14] Such nonhomogeneity could help account for the increasing relative difference in the ratio of incidence rates in Whites vs other groups as SES of census tract increased (Table 1).

It is important to attempt to define reasons why the incidence of gonorrhea is so unequally distributed and concentrated within certain core populations. Many factors clearly interact to increase the incidence of sexually transmitted diseases within such core groups.[11–13] Race and ethnicity, in particular, are best viewed not as risk factors or determinants of gonorrhea incidence, but as "risk markers" for behaviors of subgroups within racial or ethnic categories that lead to exposure to or persistence of gonorrhea. Sexual behaviors leading to exposure to gonorrhea include early onset of sexual intercourse,[8] large numbers of casual sex partners, and particularly selection of partners who are themselves members of high-risk core groups.[13] Health behaviors leading to persistence or increased transmission of gonorrhea include failure to recognize symptoms of gonorrhea, delay in seeking treatment after onset of symptoms, continued sexual activity after onset of symptoms, delay in notifying sexual partner(s) of exposure to infection, nonuse of barrier prophylaxis, and noncompliance with therapy. These behaviors in turn may be heavily influenced by high prevalence of or exposure to prostitution, illicit drug use, low economic and academic achievement (including unemployment and school dropout), nuclear family disintegration, limited access to health and behavioral intervention resources, delayed or low utilization of available health care resources, and related socioeconomic and environmental factors. Cultural perceptions of health and disease may also play a role in the uninterrupted cycle of gonorrhea transmission in such settings. Cultural factors have been poorly studied and urgently require more careful behavioral research involving population-based samples.

Overall, these data are consistent with theories that subgroups within the general population have extraordinarily high rates of gonorrhea. Individuals within these groups undoubtedly serve as a pool of high frequency transmitters of infection and play an important role in maintaining the incidence of infection in the general population. The implications of these data are that improved efforts to prevent the transmission of gonorrhea should be directed to high-risk and high-prevalence populations. These efforts may require refocusing and careful evaluation of traditional disease control measures, including culture diagnosis, effective treatment, routine screening of gonococcal isolates for antimicrobial resistance, and partner notification. Appropriate reallocation of resources for personnel and clinics in geographic areas more accessible to high-risk communities may be needed. In addition, behavioral interventions must be developed for high-incidence groups. Primary interventions clearly should be initiated no later than age 13 in order to reach teenagers who may be exposed at a very early age not only to gonorrhea, but also to other sexually transmitted diseases. Secondary behavioral interventions for high-risk individuals who present with such diseases are also needed. Development and careful evaluation of behavioral interventions based on current theoretical models of human behavior deserve the highest priority for research.

In summary, data on gonorrhea transmission and incidence within population subgroups can serve as a useful proxy for high-risk sexual behaviors that have implications for the transmission of other sexually transmitted diseases, including human immunodeficiency virus (HIV) infection. The patterns of incidence of gonorrhea observed in this study are similar to patterns of heterosexual transmission of syphilis, chancroid, and HIV, which, like gonorrhea, appear to be concentrated in urban low-SES population subgroups in the United States.[1,15] These data reinforce the need for a reassessment and refocusing of traditional disease control strategies and for development of age-specific and culturally sensitive behavioral and social intervention strategies. ☐

Acknowledgments

We thank John Milberg for mapping the incidence of reported gonorrhea by census tract; Barbara Krekeler and Roger Tulloch for assisting in collection of gonorrhea morbidity reports; laboratory personnel and disease intervention staff of the Seattle-King County

Department of Public Health; and Andrew Blair and Laurie Johnson for preparation of figures and manuscript, respectively.

The research for this paper was supported by grant AI-12192 from the National Institutes of Health and by contract D-11904D from the Centers for Disease Control, US Public Health Service.

References

1. Aral SO, Holmes KK. Epidemiology of sexual behavior and sexually transmitted diseases. In: Holmes KK, et al., eds. *Sexually Transmitted Diseases*. 2nd Ed. New York: McGraw-Hill; 1990;19–36.
2. Zaidi AA, Aral SO, Reynolds GH, et al. Gonorrhea in the United States, 1967–79. *Sex Transm Dis*. 1983;10:72–76.
3. Centers for Disease Control. *Sexually Transmitted Disease Statistics, 1987*. Issue No. 136. Atlanta, GA: CDC; 1986.
4. Centers for Disease Control. Declining rates of rectal and pharyngeal gonorrhea among males—New York City. *MMWR*. 1984;33:295–297.
5. Judson FN. Fear of AIDS and gonorrhea rates in homosexual men. *Lancet*. 1983;2:159–160.
6. US Bureau of the Census. *Current Population Reports*. Series P-25, No. 965. Population Estimates and Projections. Washington, DC: US Govt Printing Office; 1985.
7. Moore KA, Wenk D, Hofferth SL, Hayes CD. Trends in adolescent sexual and fertility behavior. In: Hofferth SL, Hayes CD, eds. *Risking the Future: Adolescent Sexuality, Pregnancy, and Childbearing*. Washington, DC: National Academy Press; 1987; 33–74.
8. Aral SO, Schaffer JE, Mosher WD, Cates W Jr. Gonorrhea rates: what denominator is most appropriate? *Am J Public Health*. 1988;78:702–703.
9. Gale JL, Hinds MW. Urethritis incidence in males in King County, Washington, 1974–1975. *Am J Public Health*. 1978;68: 20–25.
10. *1988 Population Trends for Washington State*. Office of Financial Management, Forecasting Division, State of Washington.
11. Rothenberg RB. The geography of gonorrhea. *Am J Epidemiol*. 1983;117:688–694.
12. Yorke JA, Hethcote HW, Nold A. Dynamics and control of the transmission of gonorrhea. *J Am Vener Dis Assoc*. 1978;4:51–56.
13. Rothenberg RB, Potterat BA. Temporal and social aspects of gonorrhea transmission: the force of infectivity. *Sex Transm Dis*. 1988;2:88–92.
14. Liberatos P et al. The measurement of social class in epidemiology. *Epidemiol Rev*. 1988;10:87–121.
15. Holmes KK, Kreiss JK. Heterosexual transmission of human immunodeficiency virus: overview of a neglected aspect of the AIDS epidemic. *AIDS*. 1988;1:602–610.

Most Physicians Receive Income Guarantees, Survey Confirms

The majority of physicians who accept new positions through hospital recruitment programs receive income guarantees, new survey data show. Among the 151 hospitals surveyed by Atlanta-based Jackson and Coker, 91% offer such guarantees, which include practice start-up or development costs and a minimum income level for the physician in the first year of practice.

"Income guarantees are nothing less than security blankets," said Randy Gott, Jackson and Coker's director of research. In most cases, the physician exceeds the guarantee in the first year of practice." In fact, over half of the respondents reported subsidy payments to recruited physicians of less than 25% of the guaranteed amounts.

So intense is competition for physicians among hospitals that 73% of hospitals surveyed reported that they sponsor or subsidize physician searches for existing members of their medical staff. Among the most popular forms of shared participation are reimbursement of candidates' interview expenses, fees paid to physician recruitment firms, reimbursement of candidates' relocation costs, and income guarantees.

But how hospitals choose to use these incentives depends on several factors, including the hospital's location and the physician's specialty, according to Gott. "While a hospital in a major city might entice a physician with free office rent, reimbursement of relocation expenses, and a small financial guarantee, a rural hospital might need to offer that physician a larger and more extended financial guarantee," he said. In addition, income guarantees offered will vary by specialty and will depend upon the physician's earning potential in a specific region.

Income guarantees vary in duration. Sixty-nine percent of hospitals reported that their guarantees last 1 year; and another 21% said that their guarantees last 2 years. Only 6% of hospitals surveyed offered guarantees of 6 months. Gott is convinced that hospitals will increasingly offer 2-year guarantees to physicians in highly competitive specialties where practices tend to develop more slowly.

While 74% of hospitals reported a payback provision as a part of their income guarantees, 30% of those surveyed said they would waive a portion of the amount to be paid back if the physician stayed in the practice for a designated period of time. For example, if a physician remained in the practice for 1 year beyond the guarantee period, the hospital would forgive 33% of the guarantee. But if the physician stayed for 3 years, the hospital would forgive the entire amount to be paid back.

In 33% of hospitals surveyed, physicians can pay back income guarantees by performing a variety of services, including administrative duties (67%), consulting services (30%), emergency department coverage (27%), teaching continuing medical education classes (27%), and caring for indigent patients (27%). Added Gott: "Nearly all hospitals that require a payback allow a minimum of 1 year. They want to make a payback as easy as possible."

For more information on this survey, contact Randy Gott or Becky Fitzgerald at 800/548-5393.

Public Health Intervention in a Cocaine-Related Syphilis Outbreak

Jonathan R. Hibbs, MD, and Robert A. Gunn, MD, MPH

ABSTRACT

Background. Cocaine users and prostitutes are at high risk for syphilis, but disease control is difficult among these populations. During a cocaine-related syphilis outbreak in Chester, Pennsylvania, in 1989, we conducted a control program at sites where sex and drugs were sold.

Methods. During a 2-week period, investigators recruited persons from these sites for interview, serologic testing, and empiric treatment.

Results. Among 136 persons screened, 25 (18%) had early syphilis and 26 others (19%) had recent sexual contact with early syphilis patients. All were treated at initial screening at a cost of $402 and 12 investigator hours per case, compared to $470 and 20 hours per case when treated during routine investigator activities. This program may have contributed to a short-term decline in syphilis incidence in Chester by reducing the period of infectivity of these patients.

Conclusions. Screening and empiric treatment of persons at sites where sex and drugs are sold can be useful in short-term control of cocaine-related syphilis outbreaks. (*Am J Public Health*. 1991;81:1259–1262)

Introduction

Syphilis case-finding increased in the United States during the 1980s.[1] Transmission among cocaine users and prostitutes contributes to this problem,[2–4] and the disease is difficult to control in these populations. Syphilis control depends on treatment of patients in the early stages.[5–7] The usual approach is to treat all persons with clinical or serologic evidence of infection and their sexual partners.[8]

The Pennsylvania Health Department noted increased early syphilis case-finding in southeastern Pennsylvania during 1988; one town, Chester, had a fivefold increase during 1988 compared with 1987 (Pennsylvania Department of Health, unpublished data). Because epidemiologic information identified cocaine use and prostitution as risk factors, the Health Department targeted control efforts toward locations thought to be used for the sale of sex and drugs. In contrast to the usual approach, empiric treatment was given to all persons at the time of screening.

Methods

Chester is located 20 miles south of Philadelphia. In 1980, 24% of Chester residents lived at or below the poverty level, and 46% of residents had not graduated from high school.[9] Chester's population was 53% African-American in 1980. Public facilities for sexually transmitted disease (STD) treatment in Chester included a hospital emergency room and two publicly supported STD clinics. In 1988, 9% of syphilis cases in Chester were reported from public clinics.

Primary, secondary, and early latent syphilis (referred to collectively as early syphilis) were defined using generally accepted criteria.[10] Involvement with prostitution was defined as giving or receiving money or drugs for sex within the year prior to interview. Drug use was defined as use of any illicit drug within the year prior to interview. A syphilis contact was defined as any person named as the sexual partner of an untreated early syphilis case-patient within 6 weeks before the case-patient interview.

Health providers and laboratories are required to report to the Health Department all cases of syphilis and all laboratory test results indicative of syphilis. A Health Department investigator interviews the patient. If early syphilis is confirmed, the investigator attempts to interview the patient's recent sexual partners and facilitates medical examination and treatment for them and the case-patient. Investigators are responsible for follow-up of all reportable STDs; however, during the time of the screening program, investigator activities in Chester were devoted exclusively to syphilis.

The syphilis screening and treatment program took place between 4 and 11 PM on 6 days between April 17 and 28, 1989. Persons were recruited for screening at community sites in Chester, then taken to a treatment center. Recruiting sites were

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This paper was submitted to the journal November 1, 1990, and accepted with revisions April 2, 1991.

TABLE 1—Cocaine Use, Prostitution, and Early Syphilis among Syphilis Screening Program Clients, Syphilis Intervention Program, Chester, Pa, 1989

Risk Factor	Screening Program Clients	Cases (%)	Rate Ratio (95% CI*)
Cocaine use and prostitution	15	7 (47)	4.3 (1.6–9.4)
Cocaine only	23	6 (26)	2.4 (0.8–6.3)
Prostitution only	6	2 (33)	3.1 (0.4–9.1)
Neither	92	10 (11)	1.0 (reference)
Total	136	25 (18)	

*CI=confidence interval.

TABLE 2—Cost of Syphilis Control Activities in Chester, Pa, April 15–28, 1989, Syphilis Intervention Program

	Syphilis Control Activities	
Factors	Screening and Treatment Program	Routine Investigator Activities
Investigator hours	300	415
Investigator cost	$ 6,914	$9,564
Other costs	$ 3,143	$ 300
Total costs	$10,057	$9,864
Cases treated	25	21
Hours per case treated	12	20
Cost per case treated	$ 402	$ 469

chosen based on information provided by syphilis patients that drugs or sex were sold nearby. Police agreed that they would be absent from these sites during screening. Disease investigators alerted persons frequenting these sites about the screening program several days before it took place. On 3 days, the treatment facility was the Chester State Health Center, and on 3 days it was a mobile van equipped for patient examination and moved within a block of recruiting sites.

Investigators introduced themselves to all persons found at recruiting sites and invited them to be tested and treated for syphilis. If the person agreed, the investigator transported him or her to and from the treatment site. At the treatment site, clients were interviewed, informed of potential side effects, and asked to sign a consent form allowing syphilis testing and treatment. Consenting clients had blood drawn by a nurse, were examined by a physician, and unless they denied any sexual contacts within the past year were treated for early syphilis.[11] Clients receiving penicillin were asked to remain at the treatment site 10 minutes for observation. Rapid plasma reagin (RPR) tests were performed on all blood samples. Reactive samples were analyzed with the fluorescent treponemal antibody test.

Investigator costs were calculated by adding wages, expenses, and benefits of all investigators in Chester during the screening program. In order to identify the fraction of investigator costs due to the screening program itself, the 300 investigator hours spent on the screening program were divided by the 715 investigator hours spent doing syphilis control in Chester during the weeks the screening program took place. Other screening costs included physicians' and nurses' wages, medications, phlebotomy and injection equipment, and van rental.

Routinely identified case-patients treated at public STD clinics cost the Health Department $20 each for medications and clinical fees. Routinely identified case-patients treated by private physicians cost the Health Department only investigator time; the cost of their treatment is unknown. We calculated exact 95% confidence intervals (CI) for rate ratios (RR)[12] and odds ratios (OR).[13]

Results

During the program, 136 clients were screened, of whom 80 (59%) were male and 127 (93%) were African-American. The median age of screening program clients was 20 years (range=11–52 years). The risk of syphilis was greater among female clients (15/56 [27%] vs 10/80 [13%] among male clients; rate ratio [RR]=2.3; CI=1.1–4.7) and among clients 20 years of age or older (21/71 [30%] vs 4/65 [6%] among younger clients; CI=1.7–13.3).

Among 136 persons screened, 38 (28%) reported cocaine use, and 21 (15%) reported involvement with prostitution. Clients reporting these risk factors had increased risk of syphilis (Table 1). Among 34 clients known to be case contacts, 8 (24%) had early syphilis, compared with 17 (17%) of the 102 clients not identified as case contacts (RR=1.4; CI=0.6–3.1). We treated 114 (84%) of the 136 screening program clients, including all case-patients and case contacts. No adverse drug reactions were noted.

The screening program cost the Pennsylvania Health Department $10 057, of which $6914 (69%) was investigator related (Table 2). Compared with routine investigator activities in Chester during the same period, the screening program cost fewer dollars and fewer investigator hours per case treated.

In the first 32 weeks of 1989 (January 1 through August 12), 302 early syphilis cases were diagnosed among Chester residents—1130 cases per 100 000 persons annually. Case-finding peaked in Chester during the weeks of the screening program (Figure).

Personal characteristics of case-patients diagnosed during the screening program were generally similar to those of routinely reported cases, although the screening program case-patients were slightly more likely to report cocaine use (Table 3). Early latent syphilis was more common among screening program case-patients than among routinely reported case-patients (64% vs 44%; OR=2.2, 95% CI=0.9–5.7), particularly among men (90% vs 50%; OR=9.0, 95% CI=1.2–401).

Discussion

We intervened during the peak of an urban syphilis outbreak. Routine partner notification had limited efficacy in controlling this outbreak in its early stages. Disease control was complicated by the involvement of cocaine users and prostitutes in an economically depressed area typical of urban STD transmission.[14] Multiple anonymous sexual contacts and the illicit nature of prostitution and drug use may limit the effectiveness of partner notification among such populations.[15,16] Increasing the number of investigators,[17] broadening criteria for screening,[18] and

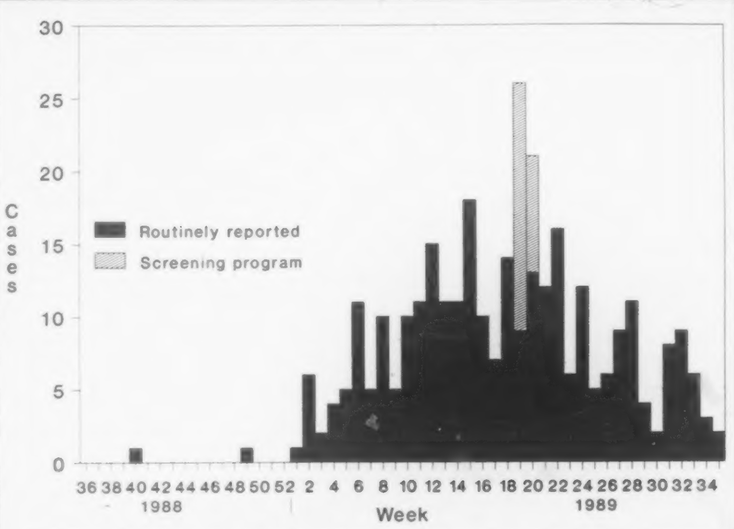

FIGURE 1—Syphilis cases by date of diagnosis and report source, Syphilis Intervention Program, Chester, Pa, 1989.

TABLE 3—Screening Program and Routinely Reported Syphilis Cases, by Age, Gender, Drug Use, and Involvement with Prostitution, Syphilis Intervention Program, Chester, Pa, 1989

	Screening Program Cases (n = 25)		Routinely Reported Cases (n = 277)[a]			
Characteristic	No.	(%)	No.	(%)	OR	(95% CI)[b]
Age <20 years	4	(16)	43	(16)	1.0	(0.3–3.3)
Female	15	(60)	157	(57)	1.2	(0.5–2.9)
Cocaine user[a]	13	(52)	82	(35)	1.7	(0.7–4.2)
Use of any illicit drug by case-patient or sexual partners[a]	16	(64)	122	(52)	1.3	(0.5–3.3)
Prostitutes and clients[a]	9	(36)	61	(26)	1.4	(0.5–3.5)

[a] Drug and prostitution information available for only 234 routinely reported cases.
[b] OR=odds ratio; CI=confidence interval.

targeting areas where high-risk persons congregate[19,20] may be indicated in STD outbreaks that routine partner notification fails to control. Our intervention combined these elements and added on-site empiric treatment.

We showed that such a program can treat infected persons and their partners in high-risk areas. In a similar screening program done during evening hours in Philadelphia, three residential sites and a soup kitchen were used. Among the 268 persons screened, 12 (4.5%) had untreated early syphilis (M. Goldberg, written communication, 1990). In a daytime outreach program conducted at recreation centers, development agencies, and a food giveaway center in Rochester, NY, 205 persons were screened and only 1 (0.5%) had untreated early syphilis (T. Huntley, written communication, 1990). A high yield of cases may depend on identifying appropriate sites and times for the screening program. We believe the training of disease investigators, their experience in Chester, and their ability to establish rapport with persons in high-risk areas also contributed to the success of our program.

Our screening program was less costly per case treated than routine investigator activities in Chester during the same period. Our estimate of routine activity costs is probably low, because we excluded the cost of private treatment for routinely identified case-patients.

This program's impact on the outbreak is difficult to substantiate. The number of cases detected per week declined after our program, suggesting an effect on incidence. It is possible that cases identified during the screening program were identified earlier than they would have been otherwise, and all the program accomplished was earlier case-finding. If that is true, the program decreased the period of infectivity of case-patients and case contacts, which should reduce STD transmission.[21] The high percentage of early latent cases (which are asymptomatic) among our male case-patients also suggests that the screening program reduced transmission, because asymptomatic men account for a disproportionate share of STD transmission[21] and are unlikely to seek medical attention on their own.

Empiric on-site treatment was a new facet of our program. Such treatment "pits the clinical importance of accurate diagnosis against the policy of public health prevention." (p. 1081)[8] Fortunately, recommended treatment of early syphilis in most patients[11] is cheap, convenient, and has few major side effects. We found empiric treatment to be cost beneficial and well tolerated. We believe that providing such treatment for high-risk populations is safe, reaches persons who may not return for follow-up, and may reduce syphilis transmission.

The mobile van in this program proved to be an effective addition to a standing permanent structure. The usefulness of this strategy is influenced by proximity to a permanent clinic and availability of an adequate, reasonably priced mobile unit. Mobile units could also improve long-term contact between health workers and the community; however, our program did not evaluate the use of mobile units for this purpose.

Since treatment does not change underlying STD risk factors,(3) interventions of this kind cannot provide long-term disease control. For short-term reductions of syphilis incidence in outbreaks associated with cocaine and prostitution, however, an approach such as ours is recommended, especially the use of empiric first-visit treatment of persons recruited for screening. Consideration should be given to using such screening for longer periods, repeating the program in the same population at intervals to determine whether a change in incidence occurs, and adding other sites. If the case yield is high, screening programs should include empiric treatment or on-site RPR screening followed by immediate therapy. Only by using a variety of strategies can the best ones be selected to combat urban syphilis outbreaks. Prompt development of such strategies is important, particularly in light of the potential for human immunodeficiency virus spread among persons with ulcerous STDs.[22] ☐

Acknowledgments

We thank Michael Cassell, Barbara Douglass, Robert Hagarty, Deborah Krasevic, Michael Lillis, Alan McNamara, Peggy Mulhollan, Edward Powers, Loretta Pursel, Nancy Breisch, RN, Carolyn Hall, RN, Robert Gens, MD, and the Chester, Pa, Board of Health, for making the Chester syphilis intervention program possible.

References

1. Centers for Disease Control. Syphilis and congenital syphilis—United States, 1985–88. *MMWR.* 1988;37:486–489.
2. Centers for Disease Control. Relationship of syphilis to drug use and prostitution. *MMWR.* 1988;37:755–759.
3. Fullilove RE, Fullilove MT, Bowser BP, Gross SA. Risk of sexually transmitted disease among black adolescent crack users in Oakland and San Francisco. *JAMA.* 1990;263:851–855.
4. Rolfs RT, Goldberg M, Sharrar RG. Risk factors for syphilis: cocaine use and prostitution. *Am J Public Health.* 1990;80:853–857.
5. Perine PL, Handsfield HH, Holmes KK, Blount JH. Epidemiology of the sexually transmitted diseases. *Annu Rev Public Health.* 1985;6:85–106.
6. Syphilis. In: Benenson AS, ed., *Control of Communicable Diseases in Man.* Washington, DC: American Public Health Association; 1985:376–380.
7. Holmes KK, Lukehart SA. Syphilis. In: Braunwald E et al., eds. *Harrison's Principles of Internal Medicine.* New York, NY: McGraw-Hill; 1987. 639–649.
8. Rothenberg RB, Potterat JJ. Strategies for management of sex partners. In: Holmes KK, Maerdh P, Sparling PF, et al., eds. *Sexually Transmitted Diseases.* 2nd ed. New York, NY: McGraw-Hill; 1990. 1081–1086.
9. *Economic and Social Indicators from the 1980 Census.* Harrisburg, Pa: Pennsylvania State Health Data Center, 1983.
10. Wharton M, Chorba TL, Vogt RL, Morse DL, Buehler JW. Case definitions for public health surveillance. *MMWR.* 1990; 39:34–35.
11. Centers for Disease Control. 1989 Sexually transmitted diseases treatment guidelines. *MMWR.* 1989;38:7–8.
12. Thomas DG, Gart JJ. A table of exact confidence limits for differences and ratios of two proportions and their odds ratios. *J Am Stat Assoc.* 1977;72:73–76.
13. Mehta CR, Patel NR, Gray R. Computing an exact confidence interval for the common odds ratio in several 2 × 2 contingency tables. *J Am Stat Assoc.* 1985;78:969–973.
14. Rothenberg RB. The geography of gonorrhea. *Am J Epidemiol.* 1983;117:688–694.
15. Andrus JK, Fleming DW, Harger DR, et al. Partner notification: can it control epidemic syphilis? *Ann Intern Med.* 1990;112:539–543.
16. Brandt AR. Sexually transmitted disease: shadow on the land, revisited. *Ann Intern Med.* 1990;112:481–482.
17. Smith WH. Blitz on syphilis in Alabama. *Public Health Rep.* 1966;81:835–841.
18. Gerber AR, King LC, Dunleavy GJ, Novick LF. An outbreak of syphilis on an Indian reservation: descriptive epidemiology and disease-control measures. *Am J Public Health.* 1989;79:83–85.
19. Potterat JJ, Rothenberg RB, Woodhouse DE, et al. Gonorrhea as a social disease. *Sex Transm Dis.* 1985;12:25–32.
20. Mellinger AK, Goldberg M, Wade A, et al. Alternative case-finding methods in a crack-related syphilis epidemic—Philadelphia. *MMWR.* 1991;40:77–80.
21. Potterat JJ, Dukes RL, Rothenberg RB. Disease transmission by heterosexual men with gonorrhea: an empiric estimate. *Sex Transm Dis.* 1987;14:107–110.
22. Quinn TC, Glasser D, Cannon RO, et al. Human immunodeficiency virus infection among patients attending clinics for sexually transmitted diseases. *N Engl J Med.* 1988;318:197–203.

Diverging Gonorrhea and Syphilis Trends in the 1980s: Are They Real?

Kenneth A. Gershman, MD, MPH, and Robert T. Rolfs, MD

ABSTRACT

Background. The purpose of this study was to evaluate whether the divergence in national trends of gonorrhea and syphilis from 1986 to 1989 in the United States was real and if overall trends masked a contemporaneous increase in both diseases in a core group.

Methods. We analyzed the following: (1) reported cases of gonorrhea and primary and secondary syphilis in the United States for the years 1981 to 1989, (2) gonorrhea screening results from six states for the years 1985 to 1989, and (3) reported cases of gonorrhea and primary and secondary syphilis by census tract for the years 1986 to 1989 in one city.

Results. The incidence of gonorrhea decreased 22% in the United States from 1986 to 1989 while the incidence of primary and secondary syphilis increased 59%. Among Blacks, syphilis incidence increased 100% and gonorrhea incidence decreased 13%; among Whites and Hispanics, the incidence of both diseases decreased. Results from gonorrhea screening among females in six states agree with gonorrhea incidence trends in those areas. Race-specific and census tract analyses of data from a number of metropolitan areas where overall rates diverged did not demonstrate a group in which the incidence of both diseases increased.

Conclusions. We conclude that diverging trends of gonorrhea and syphilis from 1986 to 1989 are real and emphasize differences in the epidemiologic characteristics of these two sexually transmitted diseases. (*Am J Public Health*. 1991;81:1263–1267)

Introduction

Declines in numbers of reported cases of gonorrhea and syphilis in the first half of the 1980s suggested that Americans had changed their sexual behavior in response to the AIDS epidemic.[1–3] Since 1986, however, syphilis incidence has increased[4] while gonorrhea incidence has continued to decline.

The recent spread of syphilis has been associated with drug use, especially cocaine.[5–8] Drug use has also been associated with the spread of gonorrhea, but this association has been less extensively studied.[9–11] Since syphilis and gonorrhea are transmitted in the same way, an increase in gonorrhea incidence, rather than a decrease, might be expected during the time when syphilis incidence increased.

The decrease in gonorrhea incidence, although probably real, could be the result of decreased surveillance and case detection activities. Anecdotally, reductions have occurred in sexually transmitted disease (STD) clinic visits in some cities and in gonorrhea screening programs in many states. Such decreases in gonorrhea case detection activities could result in artifactual declines in gonorrhea incidence. Alternatively, an overall gonorrhea decrease might mask an increase in the same core group in which syphilis has increased. To better understand if the divergence in national trends of gonorrhea and syphilis is real and whether this divergence masks a contemporaneous increase in both diseases in a subgroup of the population, we analyzed several types of data. These included (1) reported cases of gonorrhea and primary and secondary (PS) syphilis for the years 1981 to 1989, (2) gonorrhea screening results from six states for the years 1985 to 1989, and (3) reported cases of gonorrhea and PS syphilis by census tract for the years 1986 to 1989 in a city experiencing a large increase in syphilis.

Methods

We used summary data from 1981 to 1989 on cases of gonorrhea and PS syphilis reported to state health departments and sent annually to the Centers for Disease Control (CDC). Annual reports included summary data by gender, racial or ethnic group, age group, and source of report (public, private, or military) for each state and six large cities (Baltimore, Chicago, Los Angeles, New York City, Philadelphia, and San Francisco). Cases reported from public and private sources were combined; military cases were not included in these analyses. Racial or ethnic group was categorized as Black (not of Hispanic origin), White (not of Hispanic origin), Hispanic, Asian or Pacific Islander, or American Indian or Alaska Native.

Incidence rates for the United States were calculated using US Bureau of the Census population estimates for 1981–1989.[12,13] Estimates of the White and Black Hispanic populations were subtracted from estimates of Whites and Blacks to calculate estimates for Whites not of Hispanic origin and Blacks not of Hispanic origin.

Five states and two cities in 1983 and two states and three cities in 1984 did not

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This paper was submitted to the journal January 2, 1991, and accepted with revisions April 23, 1991.

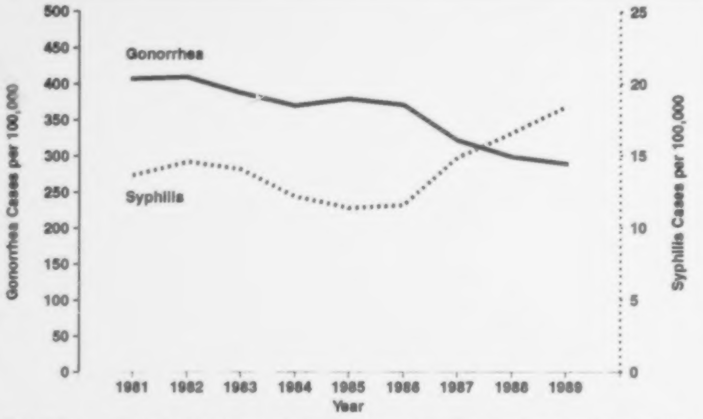

FIGURE 1—Trends in incidence of gonorrhea and primary and secondary syphilis, United States, 1981–1989.

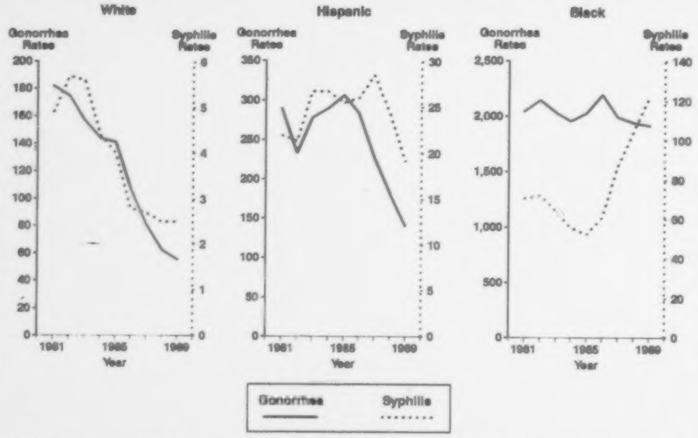

FIGURE 2—Trends in race-specific incidence of gonorrhea and primary and secondary syphilis, United States, 1981–1989. Rates = No. of cases per 100 000 persons.

submit annual reports of gonorrhea cases to CDC. Estimates for these areas were projected by averaging the sex and race distributions of cases in each area from the years before and after the missing year(s). Similar methods for interpolating missing syphilis data have been described previously.[4]

The proportion of gonorrhea cases reported with race not specified varied from year to year (17–19% for the years 1981 and 1986–1989 and 22–26% for 1982 through 1985). No cases were reported with sex not specified. Cases with race not specified were prorated to the race and sex distribution of known cases. To assess the validity of prorating, we tested varying assumptions, including the extreme assumptions that unspecified cases were all Black or all White. Gonorrhea trends were not sensitive to these extremes. Similar methods for prorating cases of syphilis with race not specified have been previously described.[4]

For the analysis of state trends, an increase in syphilis incidence was defined as a greater than 33% rate increase from 1986 to 1989 with both 1988 and 1989 rates higher than the 1986 rate. Only states with at lease 25 PS syphilis cases in 1989 were included.

Results of gonorrhea screening in women for the years 1985 to 1989 were obtained from six state STD programs where these data were available. The numbers of gonorrhea cultures performed and the numbers positive by type of facility were reported. STD clinic results were excluded and overall annual positivity rates (percent positive) were calculated for all other sources combined.

Cases of gonorrhea and PS syphilis by census tract for Rochester, NY, for the years 1986 to 1989 were obtained from the New York State STD program. Rochester was selected because gonorrhea and syphilis trends diverged there and census tract data were available. Census tracts were ranked by the number of PS syphilis cases reported in 1989. Core syphilis census tracts were defined as those with the highest ranking that cumulatively accounted for approximately 50% of cases.[14] Incidence rates were calculated using 1980 US Bureau of the Census population estimates by census tract.[15]

Results

National Trends

The incidence of both gonorrhea and syphilis decreased in the United States from 1981 to 1985 (Figure 1). From 1986 to 1989, the incidence of gonorrhea decreased 22% from a rate of 370.8 to 289.1 cases per 100 000 persons. The incidence of PS syphilis, however, increased 59% from a rate of 11.6 to 18.4 cases per 100 000 persons.

Among Whites, the incidence of gonorrhea decreased 48% from 1986 to 1989 (107.1 to 56.4 cases per 100 000), while that of syphilis decreased 11% (2.8 to 2.5 cases per 100 000) (Figure 2). Among Hispanics, the incidence of gonorrhea decreased 50% during this time (283.5 to 140.9 cases per 100 000) and syphilis incidence decreased 27% (26.0 to 19.1 cases per 100 000). However, among Blacks, a marked divergence in trends occurred: gonorrhea incidence decreased to a lesser extent than among the other groups, 13% (2197.1 to 1912.4 cases per 100 000), while syphilis incidence increased 100% (61.0 to 121.8 cases per 100 000). Sex-specific trends within each racial or ethnic group were generally similar to overall racial or ethnic trends. While syphilis incidence increased among White females from 1986 to 1989 (from 1.4 to 1.9 cases per 100 000 persons), their rates remained extremely low. Syphilis incidence among Hispanic females did not change appreciably during this time period.

Only 18 states reflect the divergence in national trends of gonorrhea and syph-

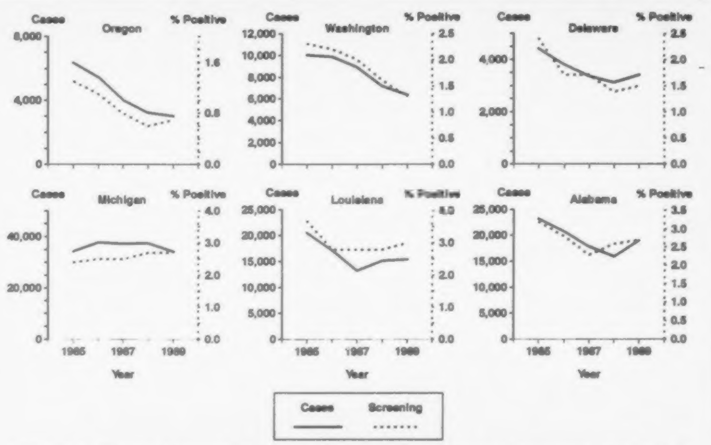

FIGURE 3—Trends in reported cases of gonorrhea and gonorrhea screening positivity rates (% positive cultures) in women, selected states, 1985–1989.

TABLE 1—Incidence of Gonorrhea and Primary and Secondary Syphilis in Core and Noncore Census Tracts in Rochester, NY, 1986–1989[a]

	Core		Noncore	
Year	Gonorrhea	Syphilis	Gonorrhea	Syphilis
1986	3721	11	1001	6
1987	3202	22	835	6
1988	4079	175	1049	35
1989	3867	266	1099	38

[a] Incidence is cases per 100 000 persons; core census tracts are those with 56% of primary and secondary syphilis cases in 1989.

ilis from 1986 to 1989. Although gonorrhea incidence rates decreased or remained unchanged in 45 states, syphilis incidence rates increased in only 22 states. In 18 of these 22 states, gonorrhea incidence decreased or remained unchanged.

Gonorrhea Screening

To evaluate whether changes in surveillance methods or case detection may account for the decrease in gonorrhea incidence, we compared trends in screening positivity rates in women and reported cases of gonorrhea in six states in which syphilis has increased (Figure 3). Screening and reported case trends were quite similar between 1985 and 1989 in these states. In two states (Oregon, Washington), screening positivity rates and reported cases both declined throughout the period 1985 to 1989; in three states (Alabama, Delaware, Louisiana), both trends initially declined then increased in parallel; and in one state (Michigan), both trends remained relatively unchanged. For two of the six states, the numbers of women screened decreased by 5% or less from 1986 to 1989. In the other four states, the numbers of women screened declined by 15% to 51%.

Core hypothesis. To evaluate whether the overall divergence in trends of gonorrhea and syphilis masked a contemporaneous increase in both diseases in a core group, we first analyzed race-specific case totals for 1986–1989 from several metropolitan areas where these data were available. In four metropolitan areas (Seattle, Memphis, Charlotte, Los Angeles) with an overall divergence in trends, stratification by race did not identify a group in which the incidence of both gonorrhea and syphilis increased.

To further evaluate the core concept, we analyzed census tract data from one metropolitan area. In Rochester, NY, gonorrhea incidence remained relatively constant from 1986 to 1989 (12% increase), while syphilis incidence increased 11-fold. We compared gonorrhea and syphilis incidence rates in the census tracts with approximately 50% of the city's reported syphilis cases (the core area) in 1989 (Table 1). Core incidence rates for syphilis and gonorrhea were 7.0 and 3.5 times higher, respectively, than noncore rates in 1989. Even in the core syphilis census tracts, however, trends for the two diseases diverged as syphilis incidence rates increased 24-fold, while gonorrhea incidence rates showed little change. A similar divergence, but of lesser magnitude, also occurred in the noncore census tracts. Of note, gonorrhea incidence rates for Rochester in 1987 were substantially lower than rates for all other years from 1984 to 1989.

Discussion

From 1986 to 1989, national trends of gonorrhea and syphilis incidence diverged: gonorrhea declined while syphilis increased markedly. This divergence occurred among Blacks, but not among Whites or Hispanics. Results from gonorrhea screening among females in a number of states support the overall decrease in gonorrhea incidence as real, rather than the result of changes in surveillance. We were unable to demonstrate a core group in which the incidence of both gonorrhea and syphilis increased.

Gonorrhea screening positivity rates declined from 1985 to 1989 in five of the six states we assessed, as did gonorrhea incidence. In the sixth state, both gonorrhea screening and incidence trends changed little. Screening positivity rates should be a reliable indicator of gonorrhea prevalence if the population screened remains relatively constant over time. For the two states in which the numbers of women screened changed little, parallel decreases in both screening and incidence trends strongly suggest that a real decrease in gonorrhea incidence occurred. In the other four states, however, the numbers of women screened decreased substantially from 1985 to 1989. Declines in screening positivity rates in these states may reflect real decreases in gonorrhea prevalence or may be the result of screening fewer high-risk women. Since STD programs are likely to reduce screening at clinics with the lowest yield and maintain screening among high-risk groups, these trends probably reflect true decreases in gonorrhea incidence.

Geographic clustering of both gonorrhea and syphilis cases among "core" census tracts in urban areas has been demonstrated previously.[14,16,17] Differential

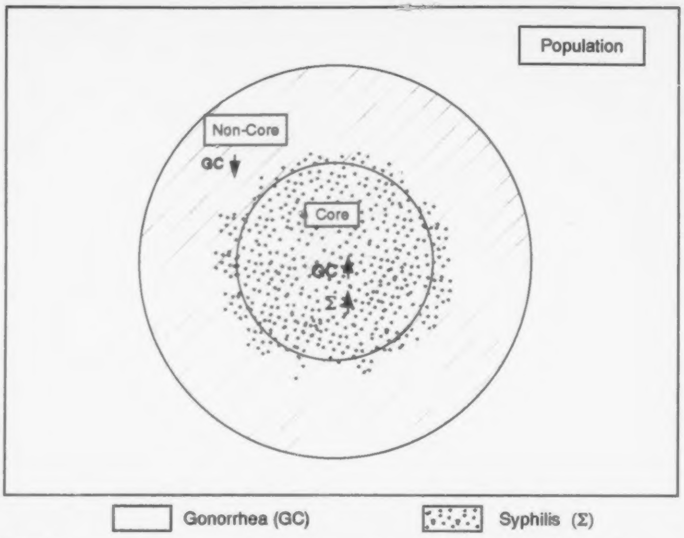

FIGURE 4—Core model of distribution of gonorrhea and syphilis in a metropolitan area.

changes in disease transmission for core and noncore groups might mask trends in the smaller core group (Figure 4). For example, the exchange of sex for money or drugs in a core group might lead to increases in both gonorrhea and syphilis incidence. Screening plus condom use or other behavior change, however, might decrease gonorrhea incidence in the much larger noncore group and mask the core group increase. Because the distribution of syphilis is largely confined to a core group, overall syphilis trends would be those of the core group.

If transmission of both infections increased in the core group while decreasing in the general population (noncore group), an overall decrease in gonorrhea incidence and increase in syphilis incidence would occur.

Neither the analysis of race-specific data in several urban areas with an overall divergence, nor the census tract analysis of another city demonstrated a group in which the incidence of both diseases increased. Race and geography are almost certainly not direct risk factors for STD; presumably they are surrogate markers for behavioral factors, which if better defined and measured would allow us to more clearly identify the core group. Race and geography alone may be inadequate to define a core group in which the incidence of both gonorrhea and syphilis increased.

It seems likely, however, that transmission of *Neisseria gonorrhoeae* has not been affected to the same extent by whatever factors have increased the spread of syphilis. Several factors might account for differences in the epidemiologic patterns of gonorrhea and syphilis in the United States. First, gonorrhea and syphilis may have different core groups. Although studies suggest that the core group involved in the recent syphilis increase is the same as the crack-using population, less evidence exists that these two groups overlap with the gonorrhea core group.[18] Second, gonorrhea and syphilis may have the same core group, but the factors that have increased the spread of syphilis may not affect the spread of gonorrhea. Oral sex has been reported to be the most frequent type of sexual activity involved in the exchange of sex for crack[19] (S. K. Koester, Ph.D., November 1990, written communication), and oral sex might be more effective in spreading syphilis than gonorrhea. Transmission of pharyngeal gonococcal infection to sex partners is thought to be inefficient and relatively rare[20] and, therefore, would be unlikely to sustain gonorrhea spread. Third, differences in health care seeking behavior among those with gonorrhea and syphilis might also contribute to the divergence. Males with gonococcal urethritis tend to seek treatment shortly after the onset of symptoms.[21] Prompt treatment translates into a short infectious period and interrupted transmission. In contrast, a large proportion of persons with infectious syphilis do not seek medical attention despite signs and symptoms.[22] Crack use and related factors such as unemployment and poverty may contribute to delays in seeking health care among persons with syphilis.

From 1986 to 1989, gonorrhea and syphilis trends have diverged for Blacks, among whom syphilis has increased dramatically since 1986, but not for Whites or Hispanics. An understanding of why syphilis but not gonorrhea incidence has increased in this population will better define transmission dynamics of these two diseases and, therefore, contribute to more successful control strategies. ☐

Acknowledgments

The authors wish to thank Annette Ladan for computer programming assistance, Laurie Kamimoto for assistance with the analysis of census tract data, and Dr. Richard B. Rothenberg for his critical review of the manuscript.

This paper was presented at the 118th Annual Meeting of the American Public Health Association, New York, NY, September/October 1990.

References

1. Centers for Disease Control. Declining rates of rectal and pharyngeal gonorrhea among males—New York City. *MMWR*. 1984;33:295–297.
2. Handsfield HH. Decreasing incidence of gonorrhea in homosexually active men—minimal effect on risk of AIDS. *West J Med.* 1985;143:469–470.
3. Judson FN. Fear of AIDS and gonorrhea rates in homosexual men. *Lancet*. 1983;2:159–160.
4. Rolfs RT, Nakashima AK. Epidemiology of primary and secondary syphilis in the United States, 1981–89. *JAMA*. 1990; 264:1432–1437.
5. Rolfs RT, Goldberg M, Sharrar RG. Risk factors for syphilis: cocaine use and prostitution. *Am J Public Health*. 1990;80:853–857.
6. Andrus JK, Fleming DW, Harger DR, et al. Partner notification: can it control epidemic syphilis? *Ann Intern Med*. 1990;112:539–543.
7. Centers for Disease Control. Relationship of syphilis to drug use and prostitution—Connecticut and Philadelphia, Pennsylvania. *MMWR*. 1988;37:755–758.
8. Centers for Disease Control. Congenital syphilis—New York City, 1986–1988. *MMWR*. 1989;48:825–829.
9. Hook EW, Brady WE, Reichart CA, Upchurch DM, Sherman LA, Wasserheit JN. Determinants of emergence of antibiotic-resistant *Neisseria gonorrhoeae*. *J Infect Dis*. 1989;159:900–907.
10. Handsfield HH, Rice RJ, Roberts MC, Holmes KK. Localized outbreak of penicillinase-producing *Neisseria gonorrhoeae*: paradigm for introduction and spread of gonorrhea in a community. *JAMA*. 1989;261:2357–2361.
11. Schwarcz, SK, Bolan GA, Fullilove MT, McCright J, Kohn R. Crack cocaine as a risk factor for gonorrhea among black teenagers. Presented at the 117th Annual Meet-

ing of the American Public Health Association, Chicago, October 22–26, 1989.

12. US Bureau of the Census. *United States Population Estimates by Age, Sex, and Race: 1980–1988*. Washington, DC: US Dept of Commerce 1990;2:55–60. Series P-25, No. 1045.
13. US Bureau of the Census. *United States Population Estimates by Age, Sex, and Race: 1989*. Washington, DC: US Dept of Commerce 1990;3:12–15. Series P-25, No. 1057.
14. Rothenberg RB. The geography of gonorrhea: empirical demonstration of core group transmission. *Am J Epidemiol*. 1983;117:688–694.
15. US Bureau of the Census. *1980 Census of Population and Housing: census tracts, Rochester, N.Y., Standard Metropolitan Statistical Area*. (PHC80-2-306) Washington, DC: US Dept of Commerce; 1983:4–11.
16. Potterat JJ, Rothenberg RB, Woodhouse DE, Muth JB, Pratts CI, Fogle JS. Gonorrhea as a social disease. *Sex Transm Dis*. 1985;12:25–32.
17. Rothenberg R. The geography of syphilis: a demonstration of epidemiologic diversity. In: Morisset R, Kurstak E, eds. *Advances in Sexually Transmitted Diseases*. Utrecht, The Netherlands: VNU Science Press; 1986:125–133.
18. Marx R, Aral SO, Rolfs RT, Sterk CE, Kahn JG. Crack, sex, and STD. *Sex Transm Dis*. 1991;18:92–101.
19. Business Research Publications. Sex for crack: how the new prostitution affects drug abuse treatment. *Substance Abuse Rep*. 1988;19:1–3.
20. Hook EW, Handsfield HH. Gonococcal infections in the adult. In: Holmes KK, Mardh PA, Sparling PF, Wiesner PJ, eds. *Sexually Transmitted Diseases*. New York, NY: McGraw-Hill; 1990:154.
21. Jacobs NF, Kraus SJ. Gonococcal and nongonococcal urethritis in men: clinical and laboratory differentiation. *Ann Intern Med*. 1975;82:7–12.
22. Brown WJ, Donohue JF, Axnick NW, Blount JH, Ewen NH, Jones OG. *Syphilis and Other Venereal Diseases*. Cambridge, Mass: Harvard University Press; 1970:41.

Hopkins Announces New Vaccine Effective Against Life-Threatening Infection

Johns Hopkins School of Public Health researchers have proven a new Hib (Haemophilus influenzae type B) vaccine effective in preventing life-threatening infection in young children when administered at 2 and 4 months of age. The vaccine, called Hib-OMPC, was tested on Navajo children who, like some other Native American populations, are 10 to 50 times more susceptible to Hib-related disease than the general US population.

"The widespread use of Hib-OMPC vaccine could virtually eliminate *H influenzae* and its deadly effects in children around the world," said lead author Mathuram Santosham, MD, MPH, director of the Johns Hopkins Center for American Indian and Alaskan Native Health. The results of the trial are reported in the June 20 *New England Journal of Medicine*.

Infection with Hib—which is actually a bacterium, not a "flu" virus—strikes 20 000 children in the United States per year. In half the cases the infection develops into meningitis, a potentially fatal infection of tissues surrounding the brain and spinal cord. Five to 10 percent of the infected die; survivors often develop neurological disorders, including hearing loss, paralysis, and speech problems. In addition to being the leading cause of meningitis, Hib can cause pneumonia; blood poisoning; infections of the ears, joints, skin, and lining of the heart; and a potentially fatal throat infection.

"The incidence of Hib-related infection is extremely high in Native American children because this population has a lower antibody response to Hib vaccines than the general population. We learned these differences in a recent study on Apache children," Santosham said.

"Proving the efficacy of Hib-OMPC vaccine in this high-risk population should also predict its efficacy in other populations," he added.

With the help of Raymond Reid, MD, MPH, a Hopkins research associate who is a Navajo physician, and a number of other investigators, Santosham directed the double blind placebo-controlled clinical trial in 3486 Navajo infants from July 1988 to August 1990 in Whiteriver, Az. The Hib vaccine was shown to be effective in preventing *H influenzae* disease when two doses were administered at 2-month intervals beginning at 6 to 12 weeks of age. When given at this early age, a single dose protected infants for at least 2 months; two doses gave protection at least to the age of 18 months. After that, a booster can be given, but the Hopkins researchers believe that generally the child's immune system by then has matured sufficiently to fight the Hib organism.

"Early vaccination is vital to the success of Hib-OMPC," said Santosham.

"We proved that this is the only vaccine available today which can be safely administered to an infant only 6 weeks old," Santosham said, "and which provides protection beginning with the first dose."

The vaccine has been developed by Merck Sharp & Dohme, who supported the research.

A Multistate Outbreak of Hepatitis A Caused by the Consumption of Raw Oysters

Jean-Claude A. Desenclos, MD, Karl C. Klontz, MD, MPH, Michael H. Wilder, MD, MS, Omana V. Nainan, MS, Harold S. Margolis, MD, and Robert A. Gunn, MD, MPH

ABSTRACT

Background. In August 1988 we investigated a multistate outbreak of hepatitis A caused by Panama City, Florida, raw oysters.

Methods. Cases of hepatitis A (HA) with onset in July–August 1988 were identified among persons who ate seafoods harvested in the coastal waters of Panama City, Florida. We conducted a case-control study, using eating companions of case-patients, and calculated attack rate (AR) per 1000 dozen raw oysters served. Enzyme immunoassay (EIA) and a polymerase chain reaction (PCR) technique were performed on samples of raw shellfish obtained from Panama City coastal waters.

Results. Sixty-one case-patients were identified in five states: Alabama (23), Georgia (18), Florida (18), Tennessee (1), and Hawaii (1). We found an increased risk of HA for raw oyster eaters (odds ratio = 24.0; 95% confidence interval = 5.4–215.0; $P < .001$). The AR of HA in seafood establishments was 1.9/1000 dozen raw oysters served. The EIA and PCR revealed HA virus antigen and nucleic acid in oysters from both unapproved and approved oyster beds, in confiscated illegally harvested oysters, and in scallops from an approved area.

Conclusions. The monitoring of coastal waters and the enforcement of shellfish harvesting regulations were not adequate to protect raw oyster consumers. More emphasis should be placed on increasing public awareness of health hazards associated with eating raw shellfish. (*Am J Public Health.* 1991;81:1268–1272)

Introduction

The consumption of raw or undercooked shellfish has caused numerous outbreaks of bacterial and viral enteric diseases worldwide.[1–18] In the United States, outbreaks of hepatitis A (HA)[4–8] and of Norwalk gastroenteritis[15–17] caused by the consumption of raw shellfish have been reported regularly since 1962, and raw or undercooked clams or oysters were implicated as the most frequent vehicle of infection.[4–8,13–17] To prevent the occurrence of such outbreaks, many states have adopted regulations to prevent the harvesting and distribution of shellfish contaminated by human fecal pathogens.[19] However, these regulations have not prevented raw shellfish–borne outbreaks of HA[8] and Norwalk virus gastroenteritis.[16,17] These outbreaks were linked to shellfish by epidemiologic data; however, to our knowledge hepatitis A virus (HAV) has not been identified in implicated shellfish during outbreaks in the United States.

We describe a large multistate outbreak of HA caused by the consumption of Florida raw oysters. During this outbreak we were able to identify HAV in illegally harvested oysters that we believe were the source of infection, using an enzyme immunoassay (EIA) and in vitro nucleic acid amplification, a method potentially applicable to monitoring the microbiological suitability of shellfish intended for human consumption.

Methods

Background

In early August 1988, cases of HA were reported among Alabama patrons of Panama City, Florida, seafood restaurants and oyster bars. Panama City, located on the Gulf of Mexico, is surrounded by three bays (East, West, and North Bay) where shellfish, particularly oysters, grow naturally. Registered fishermen harvest oysters according to a management plan monitored by the Florida Department of Natural Resources. Oyster beds are classified as approved, conditionally approved, or prohibited for harvesting according to factors such as rainfall, water salinity, and water coliform counts. For 7 years before 1988, the bays surrounding Panama City were closed to oyster harvesting each year from June 1 to August 31. However, in 1988, summer harvesting was permitted in North Bay.

Epidemiologic Investigation

A case of HA was defined as a person who was IgM antibody positive to HAV, or who was diagnosed with HA by a physician during July or August 1988, and

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This paper was submitted to the journal January 24, 1991, and accepted with revisions April 10, 1991.

Note. The inclusion of trade names in this article is for identification only and does not imply endorsement by the Department of Health and Human Services or the US Public Health Service.

who had attended a seafood event 10–50 days before symptom onset. Cases were ascertained by contacting neighboring states, all local Florida health departments, and Panama City hospitals and physicians, and through interviews with case-patients and managers of seafood establishments.

We conducted a case-control study in August 1988. All case-patients reached by telephone were included in the case group. The non-ill meal companions named by each patient (0 to 3 companions named per patient) were included in the control group if they did not develop any symptom of HA during the 10- to 50-day period following the seafood meal, if they did not report a past history of HA or jaundice, and if they had not received immune globulin during the previous 3 months. Serum specimens were not obtained to detect asymptomatic infection among controls. A standardized questionnaire was used for telephone interviews with cases and controls to ascertain health status and food consumption during the seafood event.

The direct and indirect costs of the outbreak were estimated from medical information obtained during the case-control study. Direct cost was calculated using mean routine medical fees at local hospitals; indirect cost was calculated at a mean salary of $100 per day of work lost.

To estimate attack rates (ARs) for oyster bar patrons, we reviewed oyster invoices and records in Panama City seafood establishments where patients had eaten. ARs were calculated per 1000 dozen raw oysters served by week and by establishment.

Crude odds ratios (OR) and 95% exact confidence intervals (CI)[20] were calculated for each food and exposure investigated. Statistical tests used included χ^2 with Yates correction, the two-tailed Fisher exact test, the χ^2 for trend, and the Student t test for continuous variables. When case-patients had eaten in more than one restaurant during the exposure period, the analysis of data was restricted to the restaurant which had reported other case(s) being exposed on the same day.

Environmental Investigation

We traced the origin of oysters by reviewing oyster bag tag numbers recorded by seafood establishments. Information on illegal oyster harvesting activities in Panama City coastal waters was obtained from the Florida Marine Patrol. Coliform count records from shellfish growing areas and at a sewage treatment plant (plant A) were reviewed. We inspected Panama City approved and unapproved oyster growing areas to search for sources of fecal pollution and collected samples of oysters and water from approved (North Bay) and two unapproved areas (Watson and Beatty Bayou), scallops from a scallop growing area (Shell Island), and sludge from another sewage treatment plant (plant B) located in Watson Bayou. We also obtained frozen raw oysters that had been illegally harvested in Watson Bayou in early August 1988 and confiscated by the Florida Marine Patrol.

Laboratory Investigation

Coliform counts were done on water and oyster meat samples using the three tube five dilution technique.[21] For the detection of HAV antigen and nucleic acid, individual shellfish (oysters and scallops) were removed from the shell; the digestive tract, mantle, and muscle were minced; and material from two specimens was pooled, homogenized in phosphate buffer saline + 0.1M ethylene diamine tetra acetate (EDTA), and clarified by centrifugation. Separate instruments were used to prevent cross-contamination of specimens. Supernatants of the homogenated shellfish were tested for HAV antigen using a previously described EIA.[22] HAV was quantitated from a standard curve prepared from cell culture–derived HAV, strain HAS-15,[23] and diluted in oyster meat homogenate supernatant fluid from oysters not implicated in an illness outbreak. Sludge samples from the sewage treatment plant were prepared as a 10% (w/v) suspension, sonicated, and clarified by centrifugation, and the supernatant was tested for HAV antigen.

Specimens (resuspended homogenate pellets, homogenate supernatant, and sludge) were tested for HAV-RNA by in vitro nucleic acid amplification using the polymerase chain reaction (PCR). HAV-RNA was obtained by immunocapture of virus with the capture antibody used in the HAV-Ag EIA[24] followed by heating at 900°C in reverse transcriptase buffer for 3–5 minutes. The oligonucleotide primer complementary to HAV-RNA was annealed at 90°C for 3 minutes, cooled on ice, and cDNA was synthesized at 42°C for 1 hour with 200 μmoles dNTP's, 5 units AMV reverse transcriptase, and RNAscin.[25] The second primer was added and HAV-cDNA was converted to double-stranded DNA and amplified with a heat stable (Taq) polymerase in a thermal cycler for 30 cycles (DNA Thermal cycler, Perkin-Elmer Cetus, Norwalk, Conn). Amplified nucleic acid products were analyzed by electrophoresis at 10 volts/cm on a horizontal gel (Hoefer Scientific Instruments, San Francisco, Calif) in a composite agarose gel of 3% NuSieve and 1% Sekem (FMC Bioproducts, Rockland, Md) prepared in Tris-borate-EDTA buffer containing 0.5 μg/ml ethidium bromide. DNA specific for the amplified region of the HAV genome was identified by hybridization using a 32P kinase-labeled oligonucleotide probe that was internal to the two limiting primers used in the PCR.[25]

Results

Epidemiologic Investigation

Sixty-one case-patients were identified; all but two had been tested and were IgM anti-HAV positive. Of 53 patients interviewed, all but 1 stopped working while ill and 17 (32%) were hospitalized (range = 2–21 days, median = 4 days). The duration of illness ranged from 7 to 49 days (median = 21 days), and the median number of visits to a physician while ill was four per patient (range = 1–13 visits). There were no deaths; seven secondary cases occurred among household contacts of case-patients. The total cost of the outbreak was approximately $200 000: $73 000 for direct costs, i.e., medical care and the outbreak investigation, and $130 000 for indirect costs associated with lost work days.

Illness onset ranged from July 4 to August 15 (Figure 1), the exposure period from June 3 to July 21, and the incubation period from 16 to 48 days (median = 29 days). Case-patients lived in Alabama (23 persons), Georgia (18), Florida (18), Tennessee (1) and Hawaii (1). Case-patients ate in seafood restaurants or oyster bars in Panama City (53 persons, 86.9%), in a restaurant in Alabama that served oysters harvested in Panama City (3; 4.9%), and at private family gatherings in Panama City (5; 8.2%). Age of patients ranged from 8 to 60 years (median age = 31 years), and 49 (80.3%) were male.

Fifty-three case-patients and 64 controls were enrolled in the case-control study. Cases tended to be younger than controls (mean age = 27.5 and 32.2 years, respectively; $P = .02$) and were more likely to be male (81 vs 39%; $P < .001$). The risk of HA was greater for patrons who ate raw oysters than for patrons who did not (OR = 24.0; $P < .001$) (Table 1) and increased with the number of raw oysters ingested (Table 2). Of the two case-patients who did not eat raw oysters, one ate baked oysters, and the other ate raw

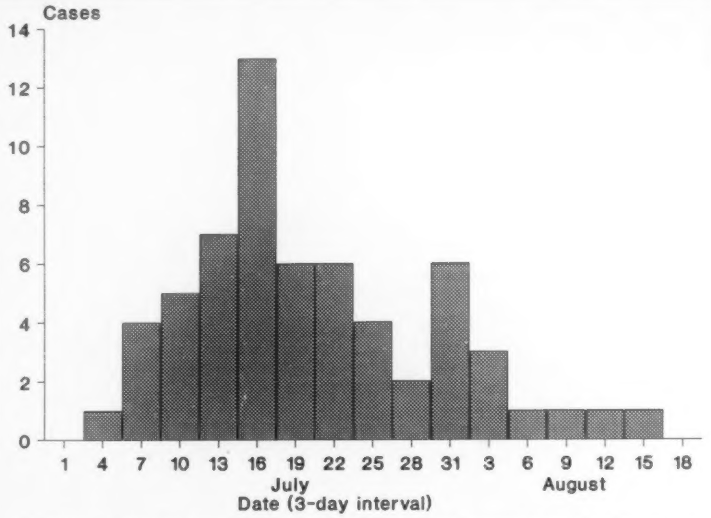

FIGURE 1—Cases of hepatitis A by date of onset, oyster associated hepatitis A outbreak, Florida, July–August 1988.

TABLE 1—Seafoods Consumed by Case-Patients and Controls, Oyster-Associated Hepatitis A Outbreak, Florida, July–August 1988

Seafood	Cases (n = 53) No.	Cases (n = 53) %	Controls (n = 64) No.	Controls (n = 64) %	Odds Ratio	95% CI[a]	P
Raw oysters	51	96	33	52	24.0	5.4–214.7	<.001
Fried or baked oysters	7	13	13	20	0.6	0.2–1.8	.4
Steamed oysters	1	2	5	8	0.2	0.01–2.1	.2
Raw shrimp	3	6	1	2	3.8	0.3–201.5	.3
Cooked shrimp	17	32	30	47	0.5	0.2–1.2	.1
Steamed clams	1	2	3	5	0.4	0.01–5.1	.6
Fish	17	32	18	28	1.2	0.5–2.9	.8

[a] CI = confidence interval.

TABLE 2—Number of Raw Oysters Eaten by Oyster-eating Case-patients and Controls, Oyster-Associated Hepatitis A Outbreak, Florida, July–August 1988

Number of Raw Oysters Eaten	Cases (n = 51) No.	Cases (n = 51) %	Controls (n = 33) No.	Controls (n = 33) %	Odds Ratio	95% CI[a]
1–6	6	12	18	55	1.0	—
7–12	20	39	11	33	5.5	1.5–21.5
>12	25	49	4	12	18.8	3.9–99.3

χ^2 for trend=20.0, $P < .001$.
[a] CI = confidence interval.

scallops caught at Shell Island beach. No other food items were associated with illness (Table 1), and the number of raw oysters eaten (>12 vs <12) had no effect on incubation period (<30 days; 55% vs 48%; rate ratio [RR] = 1.1; P = .9), duration of illness (>20 days; 79% vs 74%; RR = 1.1; P = .9), or hospitalization rate (29% vs 33%; RR = 1.1; P = .9).

During the exposure period, the 11 Panama City establishments associated with cases served approximately 27 900 dozen raw oysters. The median amount served per week was 3500 dozen (range = 1950–4800) (Figure 2), and the median number served by each establishment was 1994 dozen (range = 1050–8230). The overall AR was 1.9 cases per 1000 dozen raw oysters served with a peak during the third week of June (Figure 2). ARs by establishment ranged from 0.1 to 18 cases per 1000 dozen raw oysters, and most case-patients (44/52; 84.6%) had eaten raw oysters in one of four restaurants (A–D, Table 3).

Environmental Investigation

Seafood establishments and private individuals purchased raw oysters from wholesalers who obtained most of their oysters from Panama City–registered fishermen. Panama City bays met the defined safety standards at the time they were opened. Information gained from the Florida Marine Patrol indicated that illegal oyster harvesting at night was common during the summer of 1988, particularly in Watson Bayou where bootleggers arrested in early August had obtained multiple bags that they sold directly to some restaurants and oyster bars in Panama City.

Inspection of Watson Bayou showed failing septic tanks, boat sewage disposal, and sewage treatment plant sludge in proximity to unapproved oyster beds. Review of coliform counts at the effluent outfall of sewage treatment plant A was high [>16 000 most probable number (MPN)] from March through April 1988 and again in July, but not during May and June. This plant discharged approximately 1 mile from Watson Bayou.

Laboratory Results

Coliform counts higher than the maximum allowed were found in the water of two unapproved beds, although the oyster meat of only one of the beds was abnormal, and no human fecal pollution was detectable in the approved areas (Table 4). One of the three oyster pairs from approved beds of North Bay, all four scallops obtained from Shell Island, two oyster pairs from one of the beds sampled in Watson Bayou, and Watson Bayou oysters confiscated from a bootlegger were positive for HAV antigen by the EIA. HAV-RNA was detected by PCR in all oyster samples positive for HAV antigen by EIA (Table 4). However, two oyster samples HAV antigen negative by EIA were positive for HAV-RNA by PCR including a sample from Beatty Bayou, which was unapproved, and a sample from North Bay approved oyster beds (Table 4). Suspensions of sludge obtained from the sewage treatment plant were negative for HAV antigen and for HAV-RNA by PCR.

Discussion

This oyster-borne outbreak of HA is the largest reported in the United States since the 1973 Louisiana outbreak that involved 263 cases.[8] Although all case-patients recovered from their illness, the economic burden of the outbreak was high.

The risk of illness increased with the number of raw oysters consumed, suggesting a dose-response effect. However, there was no relationship between the quantity of raw oysters consumed and the incubation period, the duration, or the severity of illness. These findings suggest that the number of oysters eaten increased the chance of eating an infectious oyster rather than increasing the amount of virus ingested, which has been shown to decrease the incubation period.[26]

Although the case-control study showed that males had a greater risk of HA, this association is probably related to the use of eating companions of case-patients as controls: Florida adult males are more likely to eat raw oysters than females (Florida Behavioral Risk Factor Survey, Department of Health and Rehabilitative Services, unpublished data, 1988) and therefore to develop oyster-borne HA. Thus, controls as meal companions of case-patients were more likely to be females (spouse or girl friend).

Although the environmental investigation was not successful in definitively identifying the origin and the distribution of the contaminated oysters, we believe that contaminated oysters illegally harvested from unapproved waters were responsible for the vast majority of cases. However, we cannot exclude the possibility that some cases may have been caused by contaminated oysters harvested from approved beds of North Bay because both EIA and PCR identified HAV in oysters sampled there. Nevertheless, several factors strongly suggest that this outbreak was the result of bootlegging. First, because North Bay is the most common source of raw oysters served in Panama City, the observation of higher ARs of HA in four establishments suggests that more contaminated oysters were sold in those four establishments than in the other establishments. Second, according to anecdotal reports, many oysters had been illegally harvested from Watson Bayou in June and July 1988 and sold to specific seafood establishments in Panama City. Third, HAV was identified in oysters obtained directly from Watson Bayou and in oysters confiscated from a person who was arrested for illegal harvesting there. We also believe that the opening of Panama City coastal waters to summer harvesting after 7 years of summer closure facilitated the sale of the locally bootlegged oysters.

The raw scallops, eaten by one case-patient not exposed to oysters, were harvested off Shell Island where scallops were also found to contain HAV. This area is approximately 5 miles downstream from Watson Bayou, and a common source of fecal pollution may explain the contamination of shellfish from both locations.[14,15,18,27]

In the past, the isolation of viral pathogens from shellfish implicated in disease outbreaks has been difficult.[27] The finding of HAV by immunoassay in oysters obtained from the various harvesting areas indicated high levels of contamination. Even in the approved areas, oyster and scallop samples were positive for HAV by either or both EIA and PCR, which suggests that contamination may have been more widespread than was originally suspected. Virus isolation by cell culture was not attempted because of the extreme difficulty in growing wild-type HAV from environmental samples.[28] While direct proof of infectivity of either HAV-antigen or HAV-RNA positive oysters was not obtained, the epidemiologic evidence for infectivity was substantial, and it has been shown that HAV remains viable for long periods of time in water.[28]

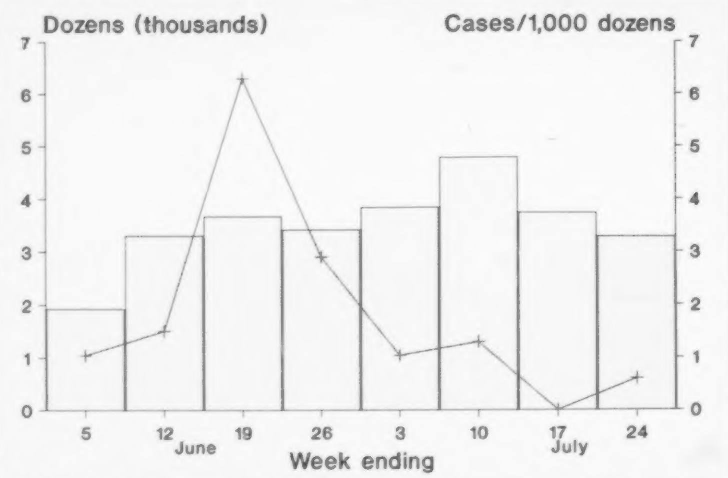

FIGURE 2—Raw oysters served (bar) and attack rate of hepatitis A per 1000 dozen raw oysters served (line) by week of exposure, oyster-associated hepatitis A outbreak, Florida, June–July 1988.

TABLE 3—Hepatitis A Attack Rate by Panama City Seafood Restaurants or Oyster-Bars[a] of Raw Oyster Consumption, Oyster-Associated Hepatitis A Outbreak, Florida, July–August 1988

Restaurant	No. of Cases	Cases per 1000 Dozen Raw Oysters
A	24	18.0
B	9	5.4
C	6	2.5
D	5	1.1
Others[b]	8	0.4
All	52	1.9

[a] n = 11.
[b] n = 7.

The application of rapid viral diagnostic methods (EIA or PCR) for the identification of HAV and presumably other viral pathogens in shellfish has the potential for assuring the quality of this raw food product. However, these methods must be verified by infectivity studies in cell cul-

TABLE 4—Coliform Counts and Hepatitis A Virus Identification in Water and Shellfish Samples, Oyster-Associated Hepatitis A Outbreak, Florida, July–August 1988

		Shellfish		
Sample	Water Coliform[a]	Coliform[b]	HAV-EIA	HAV-PCR
Approved areas				
North Bay[c]	13	<20		
Pair 1			+	+
Pair 2			−	+
Pair 3			−	+
Shell Island[d]	NA	NA	+	+
Nonapproved areas				
Watson Bayou 1°[c]	240	170	+	+
Watson Bayou 2°[c]	NA	NA	−	−
Beatty Bayou[c]	30	330	−	+
Confiscated oysters	NA	NA	+	+

Note: HAV = hepatitis A virus; EIA = enzyme immunoassay; PCR = polymerase chain reaction; + = positive test result; − = negative test result; NA = not available.

[a] Most probable number per 100 cc; maximum level permitted for water is 14 per 100 cc.

[b] Most probable number per 100 g; maximum level for shellfish is 230/100 g.

[c] Oysters.

[d] Scallops.

ture or nonhuman primates. This outbreak points out the failures of current regulations in assuring the quality of raw shellfish because enforcement cannot effectively prevent illegal harvesting, and it suggests that even approved areas may be contaminated with HAV. Emphasis must be placed on increasing the public awareness of the health risks associated with the consumption of raw or undercooked shellfish. ☐

Acknowledgments

We would like to thank the following persons for their contributions to the investigation: Charles H. Woernle, Charlotte Crysel, David Smith, Peter Sylvester, Marge MacClesky, Michael Sarra, Alan Bowen, David Heil, William Teeham, and Ernie Barnett.

References

1. Lumsden LL, Hasseltine HE, Leake JP, et al. A typhoid-fever epidemic caused by oyster-borne infection (1924–1925). *Public Health Rep.* 1925;50(suppl):1–102.
2. Morris JG, Black RE. Cholera and other vibrioses in the United States. *N Engl J Med.* 1985;312:343–350.
3. Roos B. Hepatitis epidemic transmitted by oysters. *Svenska lak-tidning.* 1956;53:989–1003.
4. Mason JO, McLean WR. Infectious hepatitis traced to the consumption of raw oysters: an epidemiologic study. *Am J Hyg.* 1962;75:90–111.
5. Dougherty WJ, Altman R. Viral hepatitis in New Jersey, 1960–61. *Am J Med.* 1962;32:704–716.
6. Dismukes EW, Bisno AL, Katz S, Johnson RF. An outbreak of gastroenteritis and infectious hepatitis attributed to raw clams. *Am J Epidemiol.* 1969;89:555–561.
7. Ruddy SJ, Johnson RF, Mosley JW, Atwater JB, Rossetti MA, Hart JC. An epidemic of clam associated hepatitis. *JAMA.* 1969;208:649–655.
8. Portnoy BL, Mackowiak PA, Caraway CT, et al. Oyster associated hepatitis: failure of shellfish certification programs to prevent outbreaks. *JAMA.* 1975;233:1065–1068.
9. Bostok AD, Mepham P, Phillips S, Skidmore S, Hambling MH. Hepatitis A infection associated with the consumption of mussels. *J Infect.* 1979;1:171–177.
10. Ohara H, Naturo H, Watanabe W, Ebisawa I. An outbreak of hepatitis A caused by the consumption of raw oysters. *J Hyg Camb.* 1983;91:163–165.
11. O'Mahony MC, Gooch CD, Smyth DA, Thrussell AJ, Bartlett CLR, Noah ND. Epidemic hepatitis A from cockles. *Lancet.* 1983;1:518–520.
12. Appleton H, Pereira MS. A possible virus aetiology in outbreaks of food-poisoning from cockles. *Lancet.* 1977;1:780–781.
13. Earampamoorthy S, Koff RS. Health Hazards of bivalve-mollusk ingestion. *Ann Intern Med.* 1975;83:107–110.
14. Vaughn JM, Landry EF, Vicale LT, Dahl MC. Isolation of naturally occurring enteroviruses from a variety of shellfish species residing in Long Island and New Jersey marine embayments. *J Food Protect.* 1980;43:95–98.
15. Eyles MJ, Davey GR, Huntley EJ. Demonstration of viral contamination of oysters responsible for an outbreak of viral gastroenteritis. *J Food Protect.* 1981;44:294–296.
16. Gunn RA, Janowski HT, Lieb S, Prather EC, Greenberg HB. Norwalk virus gastroenteritis following raw oyster consumption. *Am J Epidemiol.* 1982;115:348–351.
17. Morse DL, Guzewich JJ, Hanrahan JP, et al. Widespread outbreaks of clam and oyster associated gastroenteritis, role of Norwalk virus. *N Engl J Med.* 1985;314:678–681.
18. Heller D, Gill ON, Raynham E, Kirkland T, Zadick PM, Stanwell-Smith R. An outbreak of gastrointestinal illness associated with consumption of raw depurated oysters. *Br Med J.* 1986;292:1726–1727.
19. Dupont HL. Consumption of raw shellfish—is the risk now acceptable? *N Engl J Med.* 1985;314:707–708.
20. Metha CR, Patel NR, Gray R. Computing an exact confidence interval for the common odds ratio in several 2 × 2 contingency tables. *J Am Stat Assoc.* 1985;80:969–973.
21. Greenberg AE, Hunt DA, eds. *Laboratory Procedures for the Examination of Seawater and Shellfish.* Washington, DC: American Public Health Association; 1985.
22. Margolis HS, Nainan OV, Fields HA, et al. Characteristics of chemically aggregated IgM in an immunoglobulin class-specific immune complex assay. *J Immunol Methods.* 1987;102:23–31.
23. Robertson BH, Khanna B, Brown VK, Margolis HS. Large scale production of hepatitis A virus in cell culture: effect of type of infection on virus yield and cell integrity. *J Gen Virol.* 1988;69:2129–2134.
24. Robertson BH, Brown VK, Khanna B. Altered hepatitis A VP1 protein resulting from cell culture propagation of virus. *Virus Res.* 1989;13:207–212.
25. Margolis HS, Nainan OV. Identification of virus components in circulating immune complexes isolated during hepatitis A virus infection. *Hepatology.* 1990;11:31–37.
26. Istre GR, Hopkins RS. An outbreak of foodborne hepatitis A showing a relationship between dose and incubation period. *Am J Public Health.* 1985;75:280–281.
27. Gerba CP, Goyal SM. Detection of enteric viruses in shellfish: a review. *J Food Protect.* 1978;41:743–754.
28. Bloch AB, Stramer SL, Smith JD, et al. Recovery of hepatitis A virus from a water supply responsible for a common source outbreak of hepatitis A. *Am J Public Health.* 1990;80:428–430.

Bleach Programs for Preventing AIDS among IV Drug Users: Modeling the Impact of HIV Prevalence

ABSTRACT

Background. The growing importance of drug use as a mode of HIV transmission has led to increased attention to AIDS prevention among intravenous drug users (IVDUs). This analysis examines the effectiveness of bleach distribution, a program to prevent HIV transmission via shared needles.

Methods. We used a Markov model to assess the role of the initial HIV prevalence among drug users in determining the effectiveness of bleach programs. The model incorporates survey data on risk behaviors and published information describing HIV incubation and mortality. It predicts life expectancy for cohorts of IVDUs with and without a bleach program to estimate program effectiveness.

Results. We found that bleach programs can produce the greatest life-year savings in areas of low HIV prevalence. In the lowest prevalence scenario (0.02 initial prevalence), initiation of the program resulted in a projected savings of 2.3 life years per HIV-negative drug user, compared with 1.7 and 1.3 years under medium (0.25) and high (0.60) prevalence, respectively.

Conclusions. While bleach programs are beneficial in all groups of IVDUs, these results highlight the advantages of introducing bleach programs early, when prevalence is still comparatively low in a drug-user population. (*Am J Public Health.* 1991;81:1273–1279)

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Introduction

The proportion of AIDS cases occurring among persons whose primary risk factor is intravenous drug use has increased steadily since the beginning of the AIDS epidemic. In 1986, drug use was the primary risk factor for 16.8% of reported AIDS cases, compared to 21.5% currently.[1,2] Among new cases, the proportion is even higher.

Intravenous drug users (IVDUs) risk HIV infection from sexual contact and from sharing injection equipment—needles, syringes, or other items—with infected individuals. While protective changes in behavior have been reported among homosexuals, most IVDUs continue to place themselves at risk.[3,4] The growing importance of drug use as a mode of HIV transmission has increased attention to AIDS prevention in IVDU communities.

This analysis used a mathematical model to explore the effectiveness of one type of intervention, bleach distribution, for preventing AIDS among IVDUs. Bleach programs employ outreach workers to distribute small bottles of bleach, which IVDUs use to disinfect their injection equipment.[5] An important argument for the implementation of bleach programs in the United States has been that they are more politically feasible than needle exchange programs. However, arguments have been made for their use even where needle exchange programs are well established.[6]

Our study addressed the targeting of bleach programs, asking whether and to what extent initial HIV prevalence among IVDUs influences program effectiveness. A variety of obstacles, such as the difficulties of conducting longitudinal surveys of transient IVDU populations, prevent studies of existing programs from readily answering this question. Modeling therefore provides a practical means for studying the conditions that determine the success of bleach programs.

Methods

The model we used to predict bleach program effectiveness is a Markov (state-transition) model[7] similar to others developed to simulate the AIDS epidemic.[8-12] The model has four general states describing a hypothetical cohort of IVDUs (Figure 1). All IVDUs begin in an HIV-negative state. During each 1-year period, they face a risk of acquiring HIV infection from a needle-sharing partner and moving to HIV-positive status. Once infected, they face an annual probability of developing AIDS and then of dying from AIDS. HIV-negative and HIV-positive IVDUs die of other causes during the simulation. Death from non-AIDS causes is assumed to be negligible once an IVDU has developed AIDS.

We estimated probabilities of transition between model states from the literature, as described below. The model allows for recruitment of new HIV-negative drug users into the drug-using population. It also includes already HIV-positive

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This paper was submitted to the journal October 15, 1990, and accepted with revisions March 8, 1991.

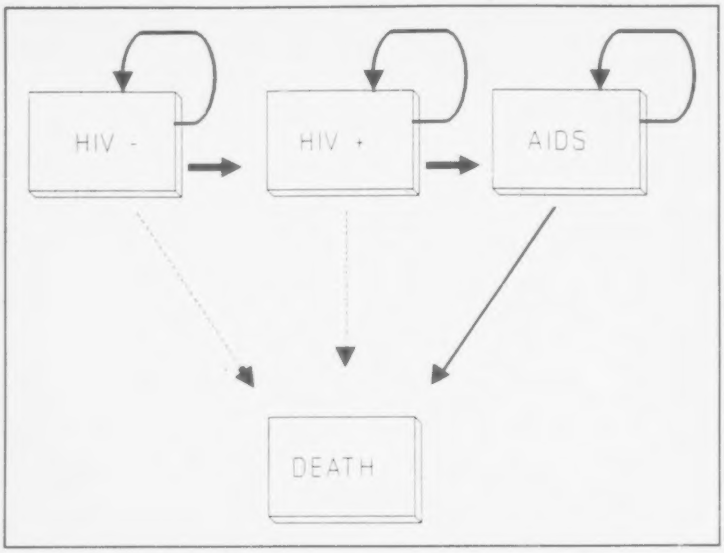

FIGURE 1—Markov Model of HIV transmission among Intravenous Drug Users.

IVDUs, who contribute to the prevalence of HIV in the community. Program effectiveness is calculated only for the initially HIV-negative IVDU cohort, however, and not for the community as a whole.

We use two measures of program effectiveness. The first, median infection-free survival, is the number of years until half of the modeled cohort become infected. The second is life expectancy, the average survival of cohort members.[7] Both measures capture the protective effect of bleach use on the individual IVDU as well as the reduction in HIV prevalence which protects other cohort members. Both also reflect continuing exposure to HIV—the fact that an IVDU who escapes infection for 1 year will be exposed again the following year. This long-term risk significantly increases lifetime probabilities of infection and is an important determinant of the effectiveness of interventions in an IVDU population.

Median infection-free survival and life expectancy are predicted both without and with a bleach program; effectiveness is defined as the improvement in these measures resulting from the program. We compared predicted effectiveness assuming programs are implemented where the HIV prevalence among drug users is low, medium, and high.

The analysis was conducted in three steps described in the results section: validation, baseline analysis, and sensitivity analysis. Change in life expectancy rather than median infection-free survival is used in the sensitivity analysis because its calculation is more precise. Computing is done on an IBM personal computer using the SMLTREE program (James P. Hollenberg, Copyright © 1985).

This model does not consider sexual transmission either within or outside of the drug-using population. While sexual transmission between individuals who also share needles is probably a secondary source of infection, sexual transmission to nondrug users is an important policy concern not directly addressed by our analysis.

Transition Probabilities

Risk of HIV infection. The cumulative annual probability of infection for IVDUs in the model depends on both personal behaviors and factors external to the IVDU (Appendix A). The risk behaviors include number of needle-sharing partners (m), injection frequency (i), and frequency of needle-sharing (s). The HIV prevalence among drug users in the community determines the probability of selecting an infected injection partner (p). A final component of risk is the probability (e) that the virus is transmitted when an IVDU shares injection equipment with an infected person. This probability is decreased when IVDUs disinfect their equipment with bleach (u,k). The model assumes that the risk of infection from each shared-needle contact with a given partner is constant and independent of previous contacts.

We used data from the Urban Health Study, a survey of IVDUs in San Francisco, to estimate drug-use behaviors.[13] From a sample of 603 interviewed in 1988, we selected those who reported both injecting drugs and sharing equipment during the previous year (n=251). The IVDUs we modeled were thus an active and at-risk subgroup of drug users.

Epidemic models suggest that heterogeneity of risk behavior may have an important effect on the course of the AIDS epidemic.[14,15] We considered heterogeneity among drug users by defining three separate groups at risk of infection. The groups are distinguished by frequency of injection (i), a behavior found to be associated with HIV infection, as our model specifies (Appendix A).[16–18] We used a partitioning algorithm, which minimizes total within-group variation, to differentiate groups within the Urban Health Study data. The resulting groups included a large number of "low-injector" IVDUs (≈1 injection per day); a "medium" group (≈4 injections per day); and a small "high" group (≈8 injections per day) (Appendix B). We determined the average proportion of injections shared (s) and number of needle-sharing partners (m) for each subgroup directly from the data.

Because injection frequency is associated with HIV infection, we assumed that the IVDU subgroups have different HIV prevalences prior to the bleach intervention (Appendix B).[16] In the course of the simulation, prevalence continued to be determined separately for the three groups. IVDUs in the model thus face a different annual probability of infection depending on their injection practices.

We estimated the probability of viral transmission per infected needle contact (e) based on the transmission in needlestick accidents among health care workers. Combining data from a number of studies, Friedland and Klein estimate a risk between 0.0013 and 0.0039 (upper confidence level = 0.0076).[19] We used 0.003 in our baseline analysis and, because this estimate is quite uncertain, a wide range was tested in the sensitivity analysis.

Bleach programs are actually multiservice outreach programs, referring IVDUs to treatment and providing information on risk behaviors. While other advantages of bleach programs may pertain, our model considers only the direct impact of bleach use on survival. We assume bleach effectiveness (k) of 85%—despite the virtually complete inactivation of HIV shown in laboratory studies of bleach[20-22]—to reflect problems in actual usage. If IVDUs disinfect only their needles, for example, the virus may be transmitted by other shared equipment. The probability that the IVDU uses bleach at each injection (u) is based on studies of IVDUs in San Francisco: 0.10 before the implementation of a program and 0.55 after.[13,23,24]

Risk of developing AIDS. Our estimates of the incubation period (time from HIV infection until the onset of AIDS) are based on data from the San Francisco Clinic Cohort, a group of HIV-positive homosexual men, because no similar studies document incubation in IVDUs.[25,26] We derive annual probabilities of developing AIDS from a Weibull model fit to these data, as described by De Gruttola and colleagues.[27,28] The Weibull model allows the estimated annual risk of developing AIDS, given HIV infection, to increase with time. The Markov model is adapted to reflect these time-dependent probabilities.

Annual probability of death. Probabilities of death for IVDUs with AIDS are based on a study of 1- to 5-year survival by Rothenberg et al.[29] Advances in treatment may ultimately increase survival, although the recent improvements among homosexual men have not been documented among IVDUs.[30] The impact of future treatments will depend on whether longer survival is associated with extended drug use. Our model currently specifies that persons with AIDS transmit HIV for only 1 year. Our conclusions would not be affected by a longer duration of infectivity, because most of the infection in the cohort takes place before AIDS develops. However, predicted life expectancy would increase. For this reason, our estimates of life expectancy are appropriate only for comparative purposes within this analysis.

Age-specific mortality from non-AIDS causes is derived from the US life tables for males[31] and adjusted to reflect higher risks of death from overdose and violence associated with drug use.[32] Not included in these probabilities is a substantial increase in mortality coincident with the AIDS epidemic, believed to be HIV-related.[33] The effect of an additional component of excess mortality among HIV-positive IVDUs is tested in the sensitivity analyses.

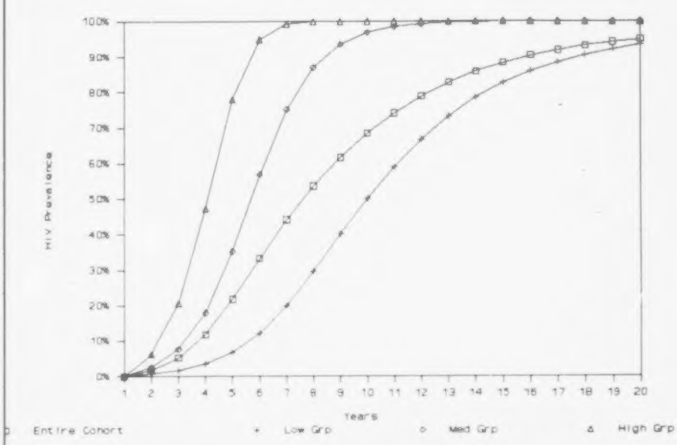

FIGURE 2—Increase in prevalence by risk group (initial prevalence 0.02).

Results

Validation: Model Dynamics and Predictions of Life Expectancy

One indicator of the validity of a model is that it obtains expected results under simple conditions. In this section, we examine how simulated life expectancy responds to variations and to extreme values of model parameters.

In the absence of AIDS, the model predicts an average undiscounted life expectancy of 26.64 years for a 35-year-old IVDU. For an IVDU who becomes infected with HIV at age 35, life expectancy is only 9.58 years. Life expectancy in the modeled cohort drops sharply when the virus is introduced, reflecting the rapid transmission of even low levels of HIV in this high-risk population.

The speed of dissemination of HIV in a drug-injecting population is illustrated by the increase in annual prevalence over time (Figure 2). When the initial HIV prevalence in the community is 2%, for example, cohort prevalence reaches 12% after 3 years and 53% after 7 years in the absence of an intervention program. (Community prevalence, which includes recruited IVDUs, is slightly lower.) This pattern of increasing annual prevalence falls within the range of trends observed in cities including New York, Edinburgh, and several Italian cities.[34-36] Prevalence increases are predictably slower among IVDUs who inject relatively infrequently and more rapid among frequent injectors.

Risk factors such as the number of injections per year (i) directly affect predicted life expectancy. Life expectancy decreases slightly at low levels of injection (Figure 3) and then more rapidly as infection becomes more probable. At high injection frequency, IVDUs are infected rapidly, and more frequent injection does not significantly worsen the risk.

While the results of these simulations are difficult to validate empirically, the model demonstrates plausible responses to changes in parameter values. Of note is the very rapid fall in life expectancy that occurs in response to modest increases in determinants of risk, such as HIV prevalence, injection frequency, or probability of viral transmission.

Baseline Estimates of Bleach Program Effectiveness

The baseline analysis examines the influence of HIV prevalence on predicted bleach program effectiveness, using our best estimates for parameter values. We compared effectiveness at three levels of overall initial HIV prevalence: low (0.02), medium (0.25), and high (0.60). These levels correspond to 1987 estimates of HIV prevalence among IVDUs in Los Angeles, Boston, and New York, respectively.[37,38]

The baseline simulations indicate that a bleach program would slow the epidemic most successfully in a low-prevalence area (Table 1). The median infection-free survival increases from 5.9 years

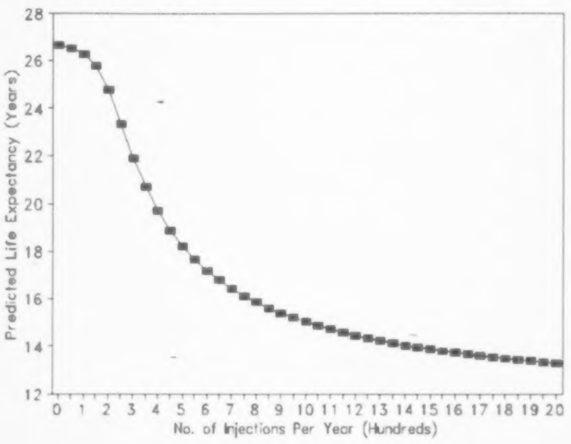

FIGURE 3—Predicted life expectancy by frequency of injection.

TABLE 1—Baseline Results for Low, Medium, and High Levels of Initial HIV Prevalence

Level	Initial Overall Prevalence[b]	Before Bleach Program	With Bleach Program	Change
Median infection-free survival, y[a]				
Low	0.02	5.9	8.6	2.7
Medium	0.25	2.9	4.4	1.5
High	0.60	1.8	2.9	1.1
Life expectancy, y				
Low	0.02	15.54	17.86	2.32
Medium	0.25	13.43	15.11	1.68
High	0.60	12.62	13.94	1.32

[a] Number of years elapsing before 50% of cohort is HIV-infected.
[b] Initial prevalence in population of cohorts and recruits; weighted average of subgroup prevalences.

TABLE 2—Gain in Life Expectancy Following Program Implementation: Sensitivity Analysis on Probability of Bleach Use

Initial Overall Prevalence	Probability of Bleach Use After Program Implemented			Complete Bleach Use and Bleach Effectiveness[b]
	55%[a]	90%	100%	
0.02	2.32	6.72	8.76	11.11
0.25	1.68	5.47	7.65	13.22
0.60	1.32	4.53	6.59	14.02

[a] Baseline value.
[b] At bleach use 100% and bleach effectiveness 100%, no further HIV infection occurs. This level of success is unlikely but illustrates the case when the greatest gain occurs in the high-prevalence scenario, where life expectancy was lowest prior to the program.

to 8.6 years. The bleach program also slows the spread of HIV in medium- and high-prevalence areas, but the change is less pronounced.

Predicted life expectancy demonstrates the same pattern of effectiveness. The bleach program adds more years to the life of an HIV-negative IVDU in a low-prevalence area than in a high-prevalence area. In the lowest-prevalence scenario, the initiation of the program results in a projected savings of 2.3 life years per HIV-negative IVDU (Table 1). The savings is 1.7 in the medium-prevalence area and 1.3 years under high prevalence. The program is thus predicted to be more effective in areas where drug users are less likely to be exposed to the virus.

Sensitivity of Results

Sensitivity analyses determine whether varying the conditions in an analysis will alter its conclusion. Changes in our model have the general effect of worsening or improving the predicted course of the AIDS epidemic. The question is therefore whether plausible changes in the predicted epidemic course would modify our basic conclusion, i.e., that bleach programs are more effective in low-prevalence areas.

Bleach use and effectiveness. Program effectiveness remains greatest in the low-prevalence scenario at any level of bleach use (u) (Table 2). This occurs because bleach effectiveness (k) is less than perfect, so that HIV infection continues even if IVDUs use bleach at all injections. Transmission is greatly slowed when both bleach use and bleach effectiveness are assumed to be higher. For example, with bleach used at 90% of injections and 95% effectiveness, median infection-free survival increases from 5.9 years without the program to over 20 years. However, combined bleach use and bleach effectiveness well over 90% would be required to change our baseline results.

Interactions among IVDUs. We examined annual recruitment of new drug users up to 10% of the original cohort size, the equivalent of, for example, an estimated 20 000 new IVDUs in New York City each year. (Table 3). Although the addition of larger numbers of uninfected IVDUs dampens the yearly increase in prevalence, higher program effectiveness is still found in the lowest prevalence area.

Sensitivity analysis on patterns of mixing among IVDU subgroups obtains a similar result. In the baseline analysis, we assumed a variant of "preferred mixing," in which IVDUs share most but not all injections within their own subgroup (Appendix B).[39] Sensitivity analyses test the effect of restricted mixing (sharing only with others in the same group) and proportional mixing (sharing unrelated to the user's own group). Effectiveness is generally higher with restricted mixing because the large low-injection subgroup is isolated from higher-prevalence IVDUs. However, overall effectiveness remains greatest at low prevalence regardless of the mixing assumption.

Components of risk: Probability of viral transmission (e) and injection frequency (i). In the sensitivity analysis on the per-injection probability of HIV transmission (e), predicted effectiveness remains highest in low-prevalence areas. However, at the lowest values of e (0.001), predicted effectiveness in low- and medium-prevalence areas is nearly the same. Effectiveness in this range of e has begun to diminish at the lowest prevalence level and would soon fall below that in medium- and high-prevalence areas. Therefore, while the baseline conclusion does not change in the sensitivity analysis, the illustration in Figure 4 reveals how still lower values of this parameter would change our results.

The sensitivity analysis on injection frequency also examines a lower risk scenario. Lower risk groups of injectors are defined by assuming five daily injections for any drug user reporting more than that number. The three risk groups are then recompiled using the method described previously. In this analysis, effectiveness increases at all prevalence areas and remains highest in the low-prevalence area.

Mortality. Although increased mortality from non-AIDS causes decreases life expectancy in our model, the pattern of program effectiveness is not changed. Effectiveness increases somewhat at all prevalence levels and remains highest at low prevalence.

TABLE 3—Gain in Life Expectancy Following Program Implementation: Sensitivity Analysis on Recruitment, Mixing, Viral Transmission, and Injection Frequency

Factors	Initial Overall Prevalence 0.02	0.25	0.60
Recruitment[a]			
No new entrants	2.11	1.52	1.22
Recruitment 5%[b]	2.32	1.68	1.32
Recruitment 10%	2.41	1.78	1.38
Mixing[c]			
Restricted	3.04	2.31	1.55
Preferred[b]	2.32	1.68	1.32
Proportionate	2.36	1.51	1.19
Probability of viral transmission per infected needle contact			
0.001	2.74	2.69	2.45
0.002	2.82	2.24	1.82
0.003[b]	2.32	1.68	1.32
0.004	1.89	1.32	1.01
0.005	1.59	1.08	0.82
Injection frequency			
Baseline[b] (max. 10/day)	2.32	1.68	1.32
Lower (max. 5/day)	2.72	1.83	1.45

[a] Recruitment is number of new HIV-negative IVDUs entering model each year as percentage of number in original HIV-negative cohort.

[b] Assumption in baseline analysis.

[c] Restricted mixing is sharing equipment only with others in the same injection-frequency group. In preferred mixing, medium- and high-injector groups share 15% of injections with a lower-risk group and remaining injections within their own group. Proportional mixing is sharing unrelated to the user's own group.

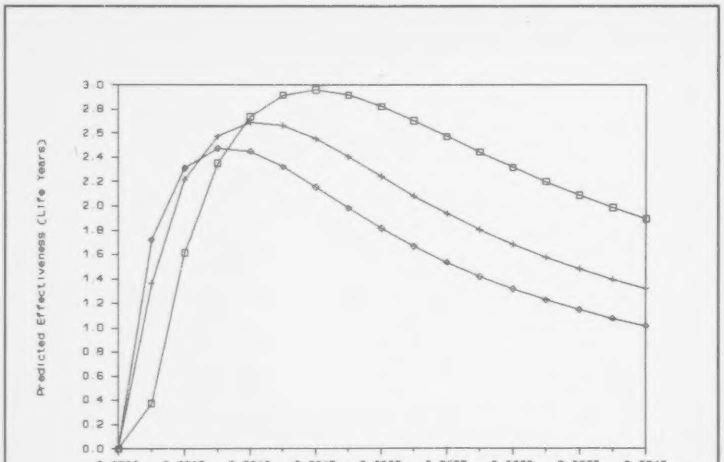

FIGURE 4—Predicted effectiveness by initial prevalence for assumptions of probability of HIV transmission per infected needle contact.

Discussion

Under our best assumptions about the AIDS epidemic among drug users, we predicted that bleach programs would be most effective in low HIV-prevalence areas. Bleach programs thus save more lives when IVDUs are less likely to be exposed to HIV. Intuitively, this occurs because IVDUs are more likely to avoid (or delay) infection in low-prevalence areas; they therefore gain more life years if bleach prevents infection at a particular point in time.

Sensitivity analyses suggest that our results are reasonably stable. Predicted effectiveness is highest in lower-prevalence areas, with two exceptions. If bleach use entirely or almost entirely eliminates HIV transmission, the program is most effective where loss of life was initially greatest—the higher-prevalence areas. Or, if HIV infection is initially extremely unlikely in the low-prevalence area, effectiveness becomes quite low because so few lives are endangered by the epidemic that bleach has few to save.

These are extreme cases, however. Generally, factors slowing the AIDS epidemic would increase, not decrease, the relative effectiveness of bleach distribution in low-prevalence areas. If IVDUs use bleach consistently and reduce risk in other ways, the combination will offer impressive life-saving potential, especially in low-prevalence areas (Table 2).

In the face of a dramatic and devastating illness such as AIDS, a reasonable response on the part of policymakers is to target resources to visibly distressed locations. What we wish to emphasize is the critical importance of preventive programs where AIDS may be less visible. It is in low-prevalence areas, before widespread infection has occurred, that bleach programs have their greatest potential for saving lives.

Several limitations should be considered before generalizing from the results of this model. There are limitations in the data, due to the intrinsic difficulty of surveying IVDUs.[40] Geographical differences in drug-use practices will affect generalizability. Some potential sources of heterogeneity have not been explored, for example, possible changes in viral infectivity over time. Our model does not reflect interruptions to drug use for periods of incarceration or drug treatment, which would lower the lifetime risk of contracting AIDS. These omissions will influence our predictions of life expectancy and may cause us to understate program benefits; they are less likely to affect our comparisons of program effectiveness across prevalence levels. As noted earlier, our analysis also excludes sexual transmission of HIV, a source of risk for IVDUs as well as their sexual partners and children. Nor are side-benefits of bleach distribution considered, such as education and referral to drug treatment.

In this analysis we have not assessed the cost or cost-effectiveness of programs in areas of differing HIV prevalence—and indeed the probable result of such an analysis is not obvious. However, because HIV prevalence among drug users can increase rapidly, our current opportunities for early intervention may be short-lived. The establishment of bleach programs in low-prevalence IVDU communities should become an explicit priority in a national effort to prevent AIDS. ☐

Acknowledgments

Supported by the National Library of Medicine Medical Informatics Research Traineeship and the National Center for Nursing Research (1 F31 NRO6341-01A1). We would like to thank Samuel R. Friedman, James P. Hollenberg, and Donald S. Shepard for their contributions. We are grateful to John K. Watters and the Urban Health Study for providing data for this project.

References

1. Centers for Disease Control. Update: acquired immunodeficiency syndrome—United States. *MMWR.* 1986;35(49):759.
2. Centers for Disease Control. *HIV/AIDS Surveillance Report.* Aug 1990, 1–18.
3. Des Jarlais DC, Friedman SR, Strug D. AIDS among intravenous drug users: A sociocultural perspective. In: Felman D, Johnson T, eds. *The Social Dimensions of AIDS: Methods and Theory.* New York, NY: Praeger; 1986;111–125.
4. Hilton B. AIDSWeek. *San Francisco Chronicle.* Jan 8 1989; A7.
5. Watters JK. Preventing human immunodeficiency virus contagion among intravenous drug users: the impact of street-based education on risk-behavior. Presented at the Third International Conference on AIDS; June 1987; Washington, DC.
6. Hartgers C, Buning EC, Coutinho RA. Evaluation of the needle exchange program in Amsterdam. Presented at the Fifth International Conference on AIDS; June 4–9 1989; Montreal, Quebec, Canada.
7. Beck RJ, Pauker SG. The Markov process in medical prognosis. *Med Decis Making.* 1983;3:419–458.
8. Anderson RM. Mathematical and statistical studies of the epidemiology of HIV. *AIDS.* 1989;3:333–346.
9. Hyman JM, Stanley EA. Using mathematical models to understand the AIDS epidemic. *Math Biosci.* 1988;90:415–473.
10. Kaplan EH. Needles that kill: modeling human immunodeficiency virus transmission via shared drug injection equipment in shooting galleries. *Rev Infect Dis.* 1989; 11:289–298.
11. Peterson D, Willard K, Altmann M, Gatewood L, Davidson G. Monte Carlo simulation of HIV infection in an IV drug user community. *J Acquir Immune Defic Syndr.* In press.
12. Halloran ME, Struchiner CJ. Prevention of AIDS in IV drug users and its consequences on the epidemic in an interacting heterosexual population. Presented at the Fifth International Conference on AIDS; June 4–9 1989; Montreal, Quebec, Canada.
13. Watters JK, Case P, Lorvick JJ, Cheng YT, Carlson J. Update on changes in HIV-1 seroprevalence and risk behavior among intravenous drug users in San Francisco. Presented at American Public Health Association Annual Meeting; Nov 1988; Boston, Mass.
14. Isham V. Mathematical modelling of the transmission dynamics of HIV infection and AIDS: a review. *J R Stat Soc.* 1989;151:5–30.
15. May RM, Anderson RM. Transmission dynamics of HIV infection. *Nature.* 1987; 326:137–142.
16. Marmor M, Des Jarlais DC, Cohen H, et al. Risk factors for infection with human immunodeficiency virus among intravenous drug abusers in New York City. *AIDS.* 1987;1:39–44.
17. Des Jarlais DC. AIDS and the sharing of equipment for illicit drug injection: a review of current data. Prepared for the National Institute on Drug Abuse by the New York State Division of Substance Abuse, New York City, 1987.
18. Schoenbaum EE, Hartel D, Selwyn PA, et al. Risk factors for human immunodeficiency virus infection in intravenous drug users. *N Engl J Med.* 1989;317:874–879.
19. Friedland GH, Klein RS. Transmission of the human immunodeficiency virus. *N Engl J Med.* 1987;317:1125–1135.
20. Newmeyer J. Why bleach? Fighting AIDS contagion among intravenous drug users: the San Francisco experience. *J Psychoactive Drugs.* 1988;20:159–163.
21. Froner G. Disinfection of hypodermic syringes by IV drug users. *AIDS.* 1987;1:134.
22. Froner GA, Rutherford GW, Rokeach M. Injection of sodium hypochlorite by intravenous drug users. *JAMA.* 1987;258:325 [Letter].
23. Watters JK, Case P, Lorvick JJ, Cheng YT, Carlson J. Update on changes in HIV-1 seroprevalence and risk behavior among intravenous drug users in San Francisco. Presented at the American Public Health Association Annual Meeting; Nov 1988; Boston, Mass.
24. Chaisson RE, Osmond D, Moss AR, Biernacki P, Feldman HW. HIV, bleach and needle sharing. *Lancet.* 1987;1:1430. Letter.
25. Lagakos SW, De Gruttola V. The conditional latency distribution of AIDS for persons infected by blood transfusion. *J Acquir Immune Defic Syndr.* 1989;2:84–87.
26. Lifson AR, Hessol NA, Rutherford GW, et al. The natural history of HIV infection in a cohort of homosexual and bisexual men: clinical manifestation, 1978–1989. Presented at the Fifth International Conference on AIDS; June 1989; Montreal, Quebec, Canada.
27. DeGruttola V, Lagakos SW. The value of AIDS incidence data in assessing the spread of HIV infection. *Stat Med.* 1989;8:35–43.
28. De Gruttola V, Mayer KH. Assessing and modeling heterosexual spread of the human immunodeficiency virus in the United States. *Rev Infect Dis.* 1988;10:138–150.
29. Rothenberg R, Woelfel M, Stoneburner R, Milberg J, Parker R, Truman B. Survival with the acquired immunodeficiency syndrome: experience with 5833 cases in New York City. *N Engl J Med.* 1987;317:1297–1302.
30. Harris, JE. Improved short-term survival of AIDS patients initially diagnosed with *Pneumocystis carinii* pneumonia, 1984–1987. *JAMA.* 1990;263:397–401.
31. US Department of Health and Human Services. Life Tables. *Vital Statistics of the U.S. 1985.* Vol II, Sec 6. Jan 1988. US Dept of Health and Human Services publication (PHS)88-1104.
32. Joe GW, Simpson DD. Mortality rates among opioid addicts in a longitudinal study. *Am J Public Health.* 1987;72:703–709.
33. Stoneburner RL, Del Jarlais DC, Benezra D, et al. A larger spectrum of severe HIV-1-related disease in intravenous drug users in New York City. *Science.* 1988;242:916–919.
34. Des Jarlais, DC, Friedman SR, Novick DM, et al. HIV-1 infection among intravenous drug users in Manhattan, New York City, from 1977 through 1987. *JAMA.* 1989;261:1008–1012.

35. Chaisson R, Moss A, Onishi R, Osmond D, Carlson J. HIV infection in heterosexual intravenous drug users in San Francisco. *Am J Public Health.* 1987;77:169–172.
36. Des Jarlais DC, Friedman SR, Stoneburner RL. HIV infection and intravenous drug use: critical issues in transmission dynamics, infection outcomes, and prevention. *Rev Infect Dis.* 1988;10:151–158.
37. Turner CF, Miller HG, Moses LE, eds. *AIDS: Sexual Behavior and Intravenous Drug Use.* Washington, DC: National Academy Press; 1989.
38. Lange R. National Institute on Drug Abuse, 1987.
39. Jacquez JA, Simon CP, Koopman J, Sattenspiel L, Perry T. Modeling and analyzing HIV transmission: the effect of contact patterns. *Math Biosci.* 1988;92:119–199.
40. Friedman SR, Des Jarlais DC. *Measurement of Intravenous Drug Use Behaviors That Risk HIV Transmission*: New York, NY: 1989. Narcotic and Drug Research Inc., unpublished.
41. Fineberg HV. Education to prevent AIDS: prospects and obstacles. *Science.* 1988;239:592–596.
42. Weinstein MC, Graham JD, Siegel JE, Fineberg HV. Cost-effectiveness analysis of AIDS prevention programs: Concepts, complications, and illustrations. In: Turner CF, Miller HG, Moses LE, eds. *AIDS: Sexual Behavior and Intravenous Drug Use.* Washington, DC: National Academy Press; 1989.
43. Allard R. A family of mathematical models to describe the risk of infection by a sexually transmitted agent. *Epidemiology.* 1990;1:30–33.

APPENDIX A—Risk of Infection for HIV-Negative Intravenous Drug Users[a]

$$r_j = 1 - [p((1-s_j e(1-uk))^{i_j/m_j} + (1-p)]^{m_j}$$

r_j = cumulative annual probability of infection
j = group of IVDUs
p = probability of selecting an infected injection partner (a function of prevalence in the several risk groups)[b]
s_j = probability that needle is shared; i.e., probability that someone used the needle immediately prior to this injection
e = efficiency of transmission of HIV by infected needle, per injection
u = probability that bleach is used at each injection[a]
k = effectiveness of bleach in eliminating HIV virus from a needle and syringe
m_j = number of partners per year
i_j = number of injections per year

[a] Further discussion in references 41–43.
[b] These parameters are group-specific in some model versions.

APPENDIX B—Model Specifications for Baseline and Sensitivity Analyses

	Baseline Analysis[a]			Sensitivity Analysis[b]		
Specification	IVDU			Subgroup		
Group-specific parameters	*Low*	*Medium*	*High*	*Low*	*Medium*	*High*
j Proportion of cohort in subgroup	0.60	0.31	0.09	0.43	0.35	0.22
i Number of injections per year	337	1311	3018	212	841	1723
s Fraction of injections shared	0.35	0.26	0.27	0.36	0.32	0.22
m Number of needle-sharing partners	11	13	12	10	14	11
p HIV prevalence among IVDUs						
Overall prevalence 0.02	0.012	0.031	0.031[c]	—	—	—
Overall prevalence 0.25	0.169	0.346	0.346[c]	—	—	—
Overall prevalence 0.60	0.478	0.704	0.704[c]	—	—	—
Cohort parameters						
u Per injection probability of using bleach						
Before bleach program implementation		0.10			—	
After bleach program implementation		0.55			0.56–1.00	
k Effectiveness of bleach		0.85			0.55–1.00	
e Probability of viral transmission per infected needle contact		0.003			0.001–0.005	
Other model features						
Mixing pattern among subgroups		Preferred[d]			Restricted; proportionate[e]	
Recruitment of new IVDUs into population (fraction of initial cohort population)		0.05			0.00–0.10	

[a] Values for group-specific parameters obtained from Urban Health Study data (see text).
[b] Sensitivity analysis on group-specific parameters assumes maximum of 1825 injections per year. Subgroups are redefined from data to obtain values of j, i, s, and m for sensitivity analysis (see text).
[c] Prevalences for medium- and high-injector subgroups assumed to be the same
[d] Medium- and high-injector groups share 15% of injections with lower risk group and remaining injections within their own group.
[e] Restricted mixing is sharing equipment only with others in same injection-frequency group; proportional mixing is sharing unrelated to user's own group.

Inpatient Morbidity among HIV-Infected Male Soldiers prior to Their Diagnosis of HIV Infection

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ABSTRACT

Background. **A natural history study of human immunodeficiency virus (HIV) disease was carried out among 1575 HIV-infected US Army men and 6220 demographically similar uninfected soldiers. Inpatient morbidity occurring up to 8 years prior to the date of HIV infection diagnosis among those men who became HIV infected was evaluated for both groups.**

Methods. **Incidence density rates were calculated for hospital admissions. Poisson regression was used to assess the trend in hospital admissions among those subsequently diagnosed with HIV infection. Prevalence ratios for discharge diagnoses were also calculated.**

Results. **Sixteen diagnoses/diagnosis categories occurred statistically more frequently among subsequently HIV diagnosed individuals than among those who remained uninfected. Among these were hepatitis B and abscess of anal/rectal region (6 to 8 years prior to HIV infection diagnosis); unspecified viral infection, enlarged lymph nodes, syphilis (3 to 5 years prior to HIV infection diagnosis); and diagnoses suggestive of acute retroviral syndrome (1 to 2 years prior to HIV infection diagnosis).**

Conclusions. **Data such as these may provide useful information to HIV surveillance efforts regarding patterns of morbidity experienced prior to HIV infection as well as to health care providers regarding patients at high risk for becoming infected with HIV. (*Am J Public Health.* 1991;81:1280–1284)**

Introduction

A natural history study of a large cohort of human immunodeficiency virus (HIV)-infected male soldiers and demographically similar uninfected soldiers was performed to investigate patterns of morbidity prior to and concurrent with early HIV infection. Hospital admission data during the period from HIV-infection diagnosis to 8 years prior to HIV diagnosis were analyzed to determine if the rate of hospital admissions differed for those later diagnosed with HIV infection compared to those who remained uninfected and if overall differences existed in the type and frequency of morbidity based on subsequent HIV-infection status.

Characterization of morbidity occurring around the time of HIV seroconversion is important for several reasons: (1) these signs and symptoms were not heretofore considered part of the clinical spectrum of HIV disease because they occur early in the clinical course, in some cases even before an individual suspects his or her HIV infection; (2) this information can be used by health care providers to assist in intervening early in the disease process; (3) it provides insight into the pathophysiologic processes that occur during the early stages of disease; and (4) it aids assessment of the burden and spectrum of morbidity that might be prevented by effective infection prevention programs.

Since October 1985, all military personnel on active duty and applicants for military service have undergone routine testing for HIV antibody (HIV-Ab).[1–4] Since the start of the screening program, the US Army HIV Data System (USAHDS) has archived the results of over 6 million HIV-Ab tests, approximately 2 million of them from active duty soldiers. The USAHDS maintains identifier-linked data files reflecting the dates and results of all HIV-Ab tests.

The US Army Medical Department also maintains a hospital discharge surveillance system called the Individual Patient Data System (IPDS). At the time of every discharge from an Army hospital, an entry is placed on IPDS consisting of demographic information, the primary discharge diagnosis and up to seven additional diagnoses, surgical procedures performed, and other medical care information. Thus, databases such as IPDS can be used to track hospitalized morbidity and medical care utilization over time for all beneficiaries of the Army health care system.

In addition to its ability to track morbidity among hospitalized cohorts, IPDS can be linked to other databases to track specific subcohorts of individuals. For example, when linked to the USAHDS registry of HIV seropositive individuals, IPDS provides a unique opportunity to evaluate the type and extent of inpatient morbidity in an otherwise healthy, employed HIV-infected population over an

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This paper was submitted to the journal September 20, 1990, and accepted with revisions April 2, 1991.

Note. The opinions or assertions contained in this paper are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

extended period both proximate to and remote from the time of HIV-infection diagnosis.

Methods

HIV-infected male soldiers on the USAHDS registry with HIV-infection diagnosis dates between 1986 and 1988 were included in the study. The comparison group was composed of uninfected male soldiers identified from US Army personnel data bases. Four uninfected soldiers were matched to each infected soldier on the basis of age (±2 years), race (White, non-Hispanic; Black, non-Hispanic; Hispanic; Asian; other), rank (junior enlisted; senior enlisted; warrant officer; commissioned officer), and length of military service (±5 years). The file of study subjects was linked by social security number to the IPDS for retrieval of ICD-9-CM coded discharge diagnoses. A reference date (RD) was defined for each HIV-infected individual as the earlier of the date of his first confirmed positive HIV-Ab test or the date of any hospital admission specifically coded for AIDS or HIV infection. Uninfected soldiers were assigned the same RD as the HIV-infected soldier to whom they were matched.

Hospital admissions for each individual were allocated to the year in which they occurred relative to the RD. Due to the small numbers of admissions occurring more than 8 years prior to the RD, only those that occurred from 1 to 8 years prior to the RD were used in the analysis. To eliminate bias resulting from individuals with many hospitalizations, one admission per individual per year was used in the analysis. Similarly, duplicate diagnoses in the same year were eliminated, resulting in the occurrence of any specific diagnosis only once per person per year.

Statistical Analysis

For this analysis, HIV-Ab status was considered to be a marker for past exposures that resulted in hospitalizations. Because we were interested in determining whether the hospitalization pattern of HIV-infected individuals differed from the pattern of individuals not infected prior to the infected individuals being diagnosed with HIV, we selected the outcomes of interest to be hospital admissions and unique discharge diagnoses/diagnosis types.

Hospital admission incidence rates were calculated as time-specific incidence densities (ID) and defined as the number of new admissions per 100 person-years available in each year prior to the RD. The measure of association was the incidence density ratio (IDR) calculated by the formula $IDR = ID_1/ID_2$, where subscripts indicate demographic and time-specific strata. The Poisson method and a completely categorical model allowed for the calculation of the number of hospital admissions per stratum weighted by the follow-up time (p*y) in each stratum, adjusted for age, race, and marital status.[5] Model fits were performed with the EGRET statistical package.[6] Ninety-five percent confidence intervals were calculated using the standard errors of β_k. The temporal trend associated with hospital admission incidence density ratios was determined over the 8 years prior to HIV infection diagnosis. The linear trend statistic calculated was the Poisson regression coefficient.

Prevalences (P) for unique discharge diagnoses, defined as the number of unique diagnoses of a particular type divided by the total number of diagnoses for both the HIV-infected and uninfected groups, were calculated over the combined 8-year period. Prevalence ratios (PR) for each unique discharge diagnosis defined as $PR_1 = P_{1(HIV\text{-}Ab+)}/P_{1(HIV\text{-}Ab-)}$, where subscripts indicate unique diagnosis types, were calculated for the combined 8-year period. Ninety-five percent confidence intervals were calculated using Taylor Series approximations.[7]

TABLE 1—Hospital Admissions, Person-Years, and Unadjusted Hospital Admission Incidence Density Ratios (cIDR) in Each of 8 Years Prior to HIV-Infection Diagnosis for HIV-Ab+ and Demographically Similar HIV-Ab− Individuals

Year Relative to Reference Date	No. Hospital Admissions HIV-Ab+	No. Hospital Admissions HIV-Ab−	No. Person-Years HIV-Ab+	No. Person-Years HIV-Ab−	cIDR (95% CI)[a]
−8	64	143	623	2487	1.79 (1.33–2.39)
−7	65	187	707	2830	1.39 (1.05–1.84)
−6	74	222	811	3276	1.35 (1.03–1.75)
−5	94	224	927	3731	1.69 (1.33–2.15)
−4	117	263	1063	4258	1.78 (1.43–2.21)
−3	152	330	1233	5004	1.87 (1.54–2.26)
−2	194	381	1422	5744	2.06 (1.73–2.44)
−1	269	535	1575	6220	1.98 (1.72–2.29)

[a] CI = confidence interval.

Results

A total of 1575 HIV-infected soldiers and 6220 uninfected soldiers were included in the study. The mean age for all subjects was 28 years. Thirty-four percent of subjects were White, non-Hispanic; 54% were Black, non-Hispanic; and 7% were Hispanic.

Hospital Admissions

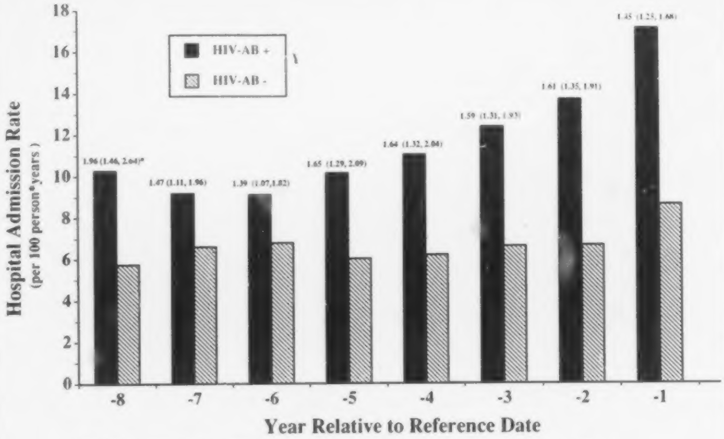
There were 4602 hospital admissions between the RD and 8 years prior for all individuals in the study, 1469 among HIV-Ab positives and 3133 among HIV-Ab negatives. After removal of duplicate admissions within each year interval, infected soldiers experienced 1029 hospital admissions while uninfected soldiers experienced 2285 hospital admissions. Table 1 presents the number of hospital admissions and available person-years for HIV-Ab positive and HIV-Ab negative individuals in each year prior to the RD.

Figure 1 presents hospital admission rates for both groups over the 8 years prior to the RD. In general, admission rates for HIV-infected soldiers displayed a monotonic increase beginning 6 years prior to HIV-infection diagnosis. In contrast, admission rates among uninfected soldiers were nearly invariant. In comparison to the combined rates during the period 6 to 8 years prior to the RD, annual hospital admission rates increased overall by 79% in HIV-infected and 34% in uninfected soldiers. Admission rates were significantly greater in all years among those who were subsequently diagnosed with HIV infection. Further, the excess in hospital admissions among those with HIV infection became greater as the RD was approached (regression coefficient t test = −0.071, $P < .001$).

Individual Diagnoses

There were 1903 and 3671 diagnoses among HIV-infected and uninfected men, respectively. Among the infected men, 792 unique diagnoses were reported. Further analysis focused on only these diagnoses. Their frequencies ranged from 1 to 74 among infected men and from 0 to 189 among those uninfected. Of the 792 diagnoses, 120 (15%) had four or more occurrences among HIV-infected soldiers. Of these 120 conditions diagnosed four or more times, 79 (66%) were more frequent (PRs >1.0) among HIV-infected vs uninfected men. Of these, 21 diagnoses were nominally statistically more common among men later diagnosed with HIV infection and had PRs ≥2.0. Table 2 presents the PRs and 95% confidence intervals for these 21 diagnoses.

Table 3 presents a qualitative description of the major causes of morbidity among individuals in this study. Those diagnoses with elevated PRs in each of three time periods are shown. Two diagnoses, hepatitis B and abscess of the anal/rectal region, are more common over the entire 8-year follow-up period among individuals subsequently diagnosed with HIV infection. From 1 to 5 years prior to HIV diagnosis, conditions associated with increased risk of HIV infection (e.g., syphilis and sexually transmitted diseases [STDs] other than syphilis) and with early HIV disease (e.g., unspecified viral infection and enlargement of lymph nodes) are significantly more common. Finally, at 1 to 2 years prior to diagnosis, conditions possibly associated with acute retroviral syndrome (e.g., unexplained fever and mononucleosis) are seen more frequently.



* Incidence Density Ratio (95% Confidence Interval) adjusted for age, race, and marital status

FIGURE 1—Hospital admission incidence density rates and adjusted ratios for each of 8 years prior to the reference date.

TABLE 2—Diagnoses with Prevalence Ratios (PR) ≥2.0 that Occur Four or More Times over the 8-Year Follow-up Period among Individuals Subsequently Diagnosed with HIV Infection

Diagnosis	No. Diagnoses HIV-Ab+	No. Diagnoses HIV-Ab−	PR	95% Confidence Interval
Syphilis[a]	42	2	40.51	9.82–167.17
Enlargement of lymph nodes	49	3	31.51	9.33–100.95
Hepatitis B[b]	37	7	10.19	4.55–22.83
Unspecified hepatitis[c]	24	11	4.21	2.07–8.57
Circumcision	5	1	9.64	1.13–82.50
Hematologic disorders[d]	24	5	9.26	3.24–24.23
Adverse drug effect[e]	10	3	6.43	1.77–23.34
Urethral scarring requiring surgery (etiology unknown)	6	2	5.79	1.17–28.65
Acute bronchitis	6	2	5.79	1.17–28.65
Sexually transmitted diseases (not including syphilis)[f]	39	16	4.70	2.63–8.39
Fever of unknown origin	9	4	4.34	1.34–14.08
Chronic tonsillitis	11	6	3.54	1.31–9.55
Unspecified viral infection	58	50	2.24	1.54–3.25
Disorders of anal/rectal region[g]	46	40	2.21	1.46–3.38
Unspecified disorders of anus/rectum/GI tract[h]	30	27	2.14	1.28–3.59
Acute pharyngitis	21	19	2.13	1.15–3.95
Herpes zoster	4	1	7.72	0.86–68.99
Hepatitis A	5	2	4.82	0.94–24.83
Viral meningitis	4	3	2.57	0.57–11.48
Syncope/collapse	8	7	2.20	0.80–6.07
Mononucleosis	9	8	2.17	0.84–5.61

[a] Latent syphilis, secondary syphilis, unspecified syphilis, neurosyphilis.
[b] Viral hepatitis B (with or without hepatic coma), chronic hepatitis, other specified viral hepatitis.
[c] Unspecified viral hepatitis, unspecified hepatitis, hepatitis.
[d] Sickle cell trait, thrombocytopenia, diseases of white blood cells, agranulocytosis, DiGeorges syndrome.
[e] Adverse effects of anti-infectives, tranquilizers, penicillin, and anti-rheumatics; erythema multiforme.
[f] Viral warts, acute gonococcal infection, unspecified urethritis, lymphogranuloma venereum, other venereal disease.
[g] Abscess of anal/rectal region, anal fissure or fistula, internal or external hemorrhoids.
[h] Hemorrhage of GI tract, idiopathic proctocolitis, unspecified disorder of rectum/anus, intestinal infection due to other organism (viral), unspecified gastritis, acute pancreatitis.

Discussion

The AIDS epidemic was first recognized in the late 1970s and early 1980s when previously rare illnesses began to be reported among gay men in San Francisco and New York.[8,9] Since that time these illnesses have been found to be associated with late-stage HIV disease. The natural history of late-stage HIV disease has been well characterized.[10-12] However, there have been relatively few detailed characterizations of the early stages of HIV disease.

Current interest in early intervention in the HIV disease course has focused interest on its presymptomatic stages.[13,14] Recent work has shown the efficacy of antiviral and prophylactic therapeutic intervention.[15,16] Trials are underway to assess effects of therapies when given in early, asymptomatic stages of HIV disease. It is important to clearly understand the early natural history of this disease before therapies are made available for general use.

It is important to document the usual course of HIV disease for a number of reasons. First, the early course is often thought of as presymptomatic. Indeed, individuals in late stages of HIV disease are likely to have trouble recalling illnesses that occurred years earlier. Furthermore, when a diagnosis is made early in the clinical course, interventions are applied that are specifically meant to delay or prevent the presentation of signs and symptoms. Medical surveillance databases that can document morbidity and HIV-infection status are not commonly available. Populations that are routinely screened for HIV and that have access to medical care in a single system are uncommon. Finally, it is difficult to determine whether morbidity that occurred years prior to a diagnosis of HIV disease was associated with the pathophysiology of HIV disease or was part of the background of morbidity of the base population.

In this study we investigated the natural history of HIV disease using routinely and systematically collected hospital admission records from members of a defined base population. This method allowed investigation of the period prior to the time of each individual's HIV-infection diagnosis. Thus, the "true" (albeit partial) natural course could be described among individuals not yet aware of and, therefore, not yet receiving care specifically for their HIV infection. Since this method does not rely on recall, it is also possible to assess HIV-related morbidity, as well as other types of morbidity, including that related to behaviors associated with HIV-infection risk. Finally, this approach provides insight into morbidity in relation to the experience of uninfected individuals from the same base population.

Our results indicate that hospitalization rates significantly increased among men who were subsequently diagnosed with HIV infection. This experience was in contrast to that of a referent group. During each of the 8 years prior to the date of initial HIV-infection diagnosis, men who became HIV infected experienced significantly higher rates of inpatient morbidity than men who did not.

This increase may be explained by (1) morbidity associated with acute HIV infection (e.g., fever, mononucleosis), (2) morbidity associated with progression of HIV disease through its clinical course, and (3) morbidity associated with behaviors that increase risk of HIV infection (e.g., STDs).

TABLE 3—Diagnoses with Elevated Prevalence Ratios (PRs) over Three Time Periods Prior to HIV-Infection Diagnosis[a]

PERIOD 1: 1 to 2 Years Prior to HIV-Infection Diagnosis	PERIOD 2: 3 to 5 Years Prior to HIV-Infection Diagnosis	PERIOD 3: 6 to 8 Years Prior to HIV-Infection Diagnosis
Hepatitis B	Hepatitis B	Hepatitis B
Abscess of anal/rectal region	Abscess of anal/rectal region	Abscess of anal/rectal region
Unspecified viral infection	Unspecified viral infection	Hepatitis A
Enlargement of lymph nodes	Enlargement of lymph nodes	
Syphilis	Syphilis	
Sexual transmitted diseases (not including syphilis)	Sexual transmitted diseases (not including syphilis)	
Chronic tonsillitis	Chronic tonsillitis	
Fever of unknown origin	Streptococcal sore throat	
Mononucleosis	Essential hypertension	
Unspecified pneumonia	Acute tonsillitis	
Acute pharyngitis	Anal fissure	
Deviated nasal septum	Lumbago	

[a] Inclusion criteria for any diagnosis into the three time periods were the occurrence of that diagnosis (1) at least three times in 1 year or (2) at least two times in 2 or more years per time period among individuals later diagnosed with HIV infection.

Since exact dates of infection with HIV are unknown, the experience of the HIV-infected group cannot be further subdivided into pre- and post-HIV-infection periods. However, since HIV infection is irreversible and antibody to HIV is persistent, an "exposure gradient" can be used to characterize their experience: at the RD, 100% were infected with HIV (by definition); at an unknown time in the past, 0% were infected with HIV; in between there is a monotonic gradient of the proportion of the group with HIV infection. While at a particular point the proportion with HIV infection is not precisely known, the experience of the HIV-Ab positive group near the RD is more associated with HIV infection than any experience more remote.

Although small sample sizes for most diagnoses did not allow for year-specific analyses, we were able to observe some trends over the 8-year period. For example, lifestyle-related morbidity (e.g., STDs, anal/rectal trauma) declined as the RD was approached. It is possible that individuals diagnosed with HIV infection became aware of their risk and changed behaviors and partner selection practices prior to diagnosis. Decreasing STD rates from the early to late 1980s have been reported,[17–20] particularly among White homosexual men. These decreasing trends have been variously ascribed to heightened awareness of and fear of contracting HIV/AIDS. In this study, even at their lowest point (1–2 years prior to HIV-infection diagnosis) lifestyle-related diagnoses occurred nearly four times more often in men subsequently diagnosed with HIV. Current studies assessing risk factors for HIV infection in recently diagnosed soldiers[21] may shed light on the relationships of STDs and other lifestyle-related morbidity on risk of HIV infection.

Overall, the amount of substance use–related morbidity was not different between HIV-Ab positive and HIV-Ab negative individuals. Alcohol abuse or dependency accounted for approximately 50% of substance use–related diagnoses in both groups. The remaining morbidity was accounted for by opioid, cannabis, cocaine, barbiturate, hallucinogen, and unspecified drug abuse. Notably, there were eight opioid-dependency diagnoses among men diagnosed with HIV infection compared to none among other men. This suggests that intravenous—but perhaps not other—drug use was a factor in these infections. Studies of behaviors among recent HIV seroconverters[21] may provide insights into trends of intravenous drug use in relation to HIV infection in military populations.

St. Louis and colleagues[22,23] recently estimated community HIV prevalence based on seroprevalences among patients admitted to hospitals for non-HIV–associated diagnoses. They concluded that hospital patients in certain geographic areas of the United States should be routinely screened for HIV infection. The results of our study may be useful in broader settings to guide the clinical judgment of care providers regarding the potential risk of HIV infection among patients. A review of patient medical history and personal practices may refine assessments of risk, encourage HIV diagnoses, and focus counseling and health education efforts in routine medical practices. As a result, HIV-directed prevention/intervention programs aimed at early stages of HIV infection could have a significantly greater impact on decreasing morbidity and health care needs than just those interventions aimed at treatment and care of end-stage HIV disease (e.g., ARC, AIDS). Thus, communities may have sufficient reason to implement comprehensive prevention programs, the impact of which might be felt prior to the time when AIDS is clinically and epidemiologically apparent.

It should not be surprising that prior history of STDs, acute viral illnesses of unknown etiology, and hepatitis B were more frequent among men subsequently diagnosed with HIV infection than others. We suggest that these diagnoses should alert primary care providers of the increased risk of HIV infection, particularly in settings such as STD, emergency, and methadone maintenance clinics. Patients with sentinel diagnoses should be screened for HIV infection, counseled regarding risks of infection and transmission, and, if negative, actively followed for reevaluation. ☐

Acknowledgments

The authors thank Clayton Best, Washington Consulting Group, Washington, DC, Mary Goldenbaum, Division of Preventive Medicine, Walter Reed Army Institute of Research (WRAIR), and Z. Frank Wann, SRA Technologies, Alexandria, Va, for assistance with data management, and Dr. Lynn Levin, Division of Preventive Medicine, WRAIR, for valuable comments on earlier versions of this paper.

References

1. Herbold JR. AIDS policy development within the Department of Defense. *Milit Med.* 1986;151:623–630.
2. Kelley PW, Takafugi ET, Tramont EC, et al. The importance of HIV infection for the military. In: Wormser GP, Stahl RE, Bottone EJ, eds. *Acquired Immune Deficiency Syndrome and Other Manifestations of HIV Infection.* Noyes Publications, Park Ridge, NJ; 1987:67–85.
3. Burke DS, Brundage JF, Herbold JR. Human immunodeficiency virus infection among civilian applicants for United States military service, October 1985 to March 1986. *N Engl J Med.* 1987;317:132–136.
4. Burke DS, Brundage JF, Redfield RR, et al. Measurement of the false positive rate in a screening program for human immunodeficiency virus infections. *N Engl J Med.* 1988;319:961–964.
5. Breslow NE, Day NE. *Statistical Methods in Cancer Research, Vol. II.* Lyon, France: International Agency for Research on Cancer; 1987.
6. Epidemiological Graphics, Estimation, and Testing (EGRET) Package, Version 0.25.01, Seattle, Wash: Statistics and Epidemiology Research Corporation.
7. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research—Principles and Quantitative Methods.* Belmont, Calif:Wadsworth, Inc; 1982.
8. Centers for Disease Control. Pneumocystis pneumonia—Los Angeles. *MMWR.* 1981;30:250–252.
9. Centers for Disease Control. Kaposi's sarcoma and pneumocystis pneumonia among homosexual men—New York City and California. *MMWR.* 1981;30:305–308.
10. Baltimore D, Feinberg MB. HIV revealed: toward a natural history of the infection. *N Engl J Med.* 1990;321:1673–1675. Editorial.
11. Levy JA. Human immunodeficiency viruses and the pathogenesis of AIDS. *JAMA.* 1989;261:2997–3006.
12. Ho DD, Pomerantz RJ, Kaplan JC. Pathogenesis of infection with human immunodeficiency virus. *N Engl J Med.* 1987;317:278–286.
13. Ruedy J, Schecter M, Montaner JSG. Zidovudine for early human immunodeficiency virus (HIV) infection: who, when, and how? *Ann Intern Med.* 1990;112:721–723. Editorial.
14. Volberding P. HIV infection as a disease: the medical indications for early diagnosis. *J Acquir Immune Defic Syndr.* 1989;2:421–425. Editorial.
15. Fischl MA, Richman DD, Grieco MH, et al. The efficacy of azidothymidine (AZT) in the treatment of patients with AIDS and AIDS-related complex: a double-blind placebo-controlled trial. *N Engl J Med.* 1987;317:185–191.
16. Fischl MA, Richman DD, Hansen, et al. The safety and efficacy of zidovudine (AZT) in the treatment of patients with mildly symptomatic human immunodeficiency virus type 1 (HIV) infection: a double-blind placebo-controlled trial. *Ann Intern Med.* 1990;112:727–737.
17. Feigal DW, Hollander H, Zeigler J, Clement M, Volberding PA. Temporal trends for past sexually transmitted diseases in men with AIDS and ARC in San Francisco. In: *Abstracts of the Fifth International Conference on AIDS.* Ottawa, Ontario, Canada: International Development Research Center; 1989;1:111.
18. Judson F, Cohn D, Douglas J. Fear of AIDS and incidence of gonorrhea, syphilis, and hepatitis B 1982–1988. In: *Abstracts of the Fifth International Conference on AIDS.* Ottawa, Ontario, Canada: International Development Research Center; 1989;1:112.
19. Mekart ML. Changing sexual behavior in the 1980's. In: *Abstracts of the Fifth International Conference on AIDS.* Ottawa, Ontario, Canada: International Development Research Center, 1989;1:113.
20. Moran JS, Peterman TA, Gershman KA, Rolfs RA, Nakashima AK, and Zaidi A. Gonorrhea and syphilis trends during the AIDS epidemic: USA. In: *Abstracts of the Sixth International Conference on AIDS,* San Francisco, Calif: University of California, San Francisco; 1990;1:315.
21. McNeil JG, Peterman T, Renzullo PO, Lasley-Bibbs V, Levin L, the Study Group. Recent HIV infection in men in the Army: a case-control study. In: *Abstracts of the Sixth International Conference on AIDS.* San Francisco, Calif: University of California, San Francisco; 1990;1:265.

Utilization of Health Services in a Cohort of Intravenous Drug Users with Known HIV-1 Serostatus

ABSTRACT

Background. Intravenous drug users (IVDUs) with human immunodeficiency virus (HIV) infection and AIDS often have no health insurance or rely on public programs to finance their health care. We examined the independent contributions of HIV serostatus, clinical symptoms, CD4 cell counts, and health insurance to utilization of health services among 1881 intravenous drug users in Baltimore, Maryland.

Methods. Participants in an ongoing natural history study of HIV were informed of HIV serostatus and seropositives were informed of CD4 cell counts; 6 months later, participants were administered a questionnaire concerning self-reported use of health services, insurance coverage, and HIV-related symptoms.

Results. Of 1881 participants, 67% had health insurance (including Medicaid), 48% had at least one outpatient visit, and 12% had at least one inpatient visit within the prior 6 months. The proportion of the study population that was HIV-1 seropositive was 32%. In multivariate analysis, the single most important predictor of health care utilization was the presence of two or more HIV-related clinical symptoms. HIV positive serostatus alone or known low CD4 counts were not significantly associated with use of health care services.

Conclusions. These data suggest that HIV seropositive IVDUs are not receiving recommended preventive care. Additional efforts will be needed to ensure that HIV-seropositive drug users participate in currently recommended protocols for early treatment of asymptomatic HIV-1 infection. (*Am J Public Health.* 1991;81:1285–1290)

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Introduction

By the end of January 1991, more than 161 000 cases of acquired immunodeficiency syndrome (AIDS) had been reported in the United States (K. Taylor, CDC AIDS monthly surveillance report, personal communication). Black and Hispanic adults account for a disproportionately large number of those infected.[1–5] The overrepresentation of minorities among those with HIV infection and AIDS is thought to be largely a consequence of intravenous (IV) drug use. It is estimated that IV drug use alone or IV drug use associated with homosexual or bisexual practices is responsible for 48% of the cumulative number of AIDS cases (K. Taylor, CDC, personal communication). One important outcome of the shifting demographics of the epidemic is that responsibility for financing and providing health care for people with human immunodeficiency virus type 1 (HIV-1) infection increasingly falls to the public sector.[6–9] Public policy will therefore play a significant role in influencing the availability of care, the timing of treatment, and the patterns of health service utilization. This research provides an initial examination of health services utilization among a population of IV drug users (IVDUs). Recent clinical research has demonstrated the important role of preventive care in delaying and mitigating the effects of opportunistic infections associated with HIV-1 disease.[10] Of concern is whether the recent advances in medical treatment, as well as the benefits of early clinical intervention, are available to the legions of IVDUs who comprise the second wave of the HIV epidemic.

The present study makes use of a data base available through an ongoing natural history study of HIV infection in IVDUs. The findings describe the relationship between health status and the utilization of medical services among IVDUs, as well as the role of health insurance in determining utilization patterns within this population.

Methods

Respondents to the health care utilization survey came from the AIDS Link to Intravenous Experience (ALIVE) Study, an ongoing longitudinal natural history study of HIV infection and IV drug use. ALIVE study participants were recruited through a number of strategies. Brochures describing the study were distributed to IVDUs who were being seen in agencies such as the Maryland Division of Parole and Probation, drug treatment programs, sexually transmitted disease clinics, emergency rooms, and homeless shelters. Also, participants were recruited via word-of-mouth. Eligibility criteria for the study included age of 18 years or older and a history of IV drug use in the previous 10 years. Individuals volunteering for the study were asked a series of questions concerning drug use history, incarcera-

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This paper was submitted to the journal October 30, 1990, and accepted with revisions April 23, 1991.

TABLE 1—Sociodemographic and Drug-Use Characteristics of 1881 Intravenous Drug Users, by HIV-1 Serostatus, Baltimore, Md

Characteristic	n	% HIV-1 Positive
Age (years)		
18–24	154	29.2
25–34	896	36.0
35–44	689	30.6
≥45	142	21.8
Race[a]		
Black	1723	34.2
Non-Black	156	12.8
Gender		
Male	1491	31.5
Female	390	36.1
Legal income[a]		
<$5000	1380	33.1
$5000–$10 000	272	26.8
>$10 000	227	34.8
Education[b]		
1–8 grade	160	24.3
9–12 grade	1458	33.1
>12 grade	262	33.6
Homeless within last year		
Yes	644	32.3
No	1237	32.5
Employed at baseline[b]		
Yes	428	28.3
No	1452	33.7
Drug use		
Current injector	1411	32.6
No use in past 6 months	470	31.9
Insurance in past 6 months		
Medicaid	1085	60.5
Private	181	9.2
No insurance	615	30.3

[a] Missing 2.
[b] Missing 1.

tion, and history of homelessness in order to obscure the true eligibility requirements. Of the individuals who came to the study, 397 were disqualified because they did not admit to IV drug use. At entry into the study, 92% of the cohort demonstrated needle track marks on the extremity used for venipuncture. Participants reported a mean of 13 years of IV drug use at time of entry into the study. Forty-two percent of study participants reported a history of drug treatment within the previous 10 years and 13% reported a drug or alcohol treatment admission within the previous 6 months. The rationale and details of the data collection have been described in detail elsewhere.[11,12]

At the initial visit, after informed consent and pretest counseling procedures, trained interviewers administered a detailed questionnaire including sections on sociodemographic information, medical history, including symptoms associated with HIV disease (Appendix A), and current and past drug use and sexual practices.

All participants had blood drawn for HIV-1 antibodies. Specimens were determined to be positive if they were reactive on two ELISA tests (Genetic Systems, Seattle, Washington) and were confirmed with Western blot assay (DuPont, Wilmington, Delaware). All screening clinic participants were given AIDS risk reduction education and appointments to return to the clinic in 3 weeks to receive HIV test results. Participants were informed of the availability of a full-time addictions counselor on staff who would facilitate referrals for drug treatment. Study participants were able to see the addictions counselor on an immediate walk-in basis.

At the return visit, all seropositive individuals and a sample of 150 HIV-negative individuals were invited to participate in the follow-up clinic, which involved semiannual visits for behavioral interviews, physical exams, and venipuncture to assess CD4 counts.[13] All other study participants were invited to continue in the screening clinic, which consisted of semiannual HIV testing and interviews. At all points in the study, health was monitored, and HIV-positive individuals were given counseling and referred to the local HIV clinic for treatment or prophylaxis when indicated by symptoms or CD4 levels.

Beginning in December 1988, health care insurance and utilization questionnaires were administered to all individuals participating in the screening or follow-up components of the ALIVE study. The data presented below describe health care utilization, demographics, health status, and health insurance coverage of a group of 1881 study subjects. The validity of self-reports of insurance status was examined by comparing self-reported Medicaid coverage and Medicaid claim files on a subsample of the study population. An 88% agreement between self-reports and Medicaid files was found.

Statistical Analysis

Our analytic point of departure is a set of descriptions of the sample in terms of their use of health care services, HIV-1 serostatus, insurance coverage, and clinical symptoms. Two measures of severity of illness, presence of two or more AIDS-related symptoms, and CD4 levels were used. The selection of stratification levels for CD4 counts was chosen to conform to old and new guidelines for clinical services. Prior to 1990, a CD4 count <200 was used as an indication for initiation of prophylactic treatment with zidovudine,[9] whereas current practices call for the use of zidovudine when CD4 levels are <500.[14] Frequency distributions of variables by HIV-1 serostatus were compared using χ^2 statistics; continuous variables were compared by subgroups using analysis of variance.[15]

The independent impacts of service utilization on the three variables of primary interest were accomplished via estimation of a probit regression for two dichotomous dependent variables: whether or not any inpatient care was used by an individual during a 6-month period and whether or not any outpatient care was received during the same period. Obtaining unbiased estimates of the impact of insurance coverage on use of services is complicated because there may be unmeasured factors that determine both insurance coverage and service utilization, for example, the degree of concern with one's health. We used an instrumental variables estimation technique described by Maddala[16] to address this potential source of bias. The standard errors of the estimates for this two-stage instrumental variables model were calculated by following the procedure proposed by Amemiya.[17]

Results

Descriptive Findings

Table 1 presents sociodemographic features of participants in the health utilization study (N = 1881). This group was predominately male (79%), non-White (92%), of low socioeconomic status (income below $5000—73%), and had a median age of 34. The proportion of the study population that was HIV-1 seropositive was 32% (n = 610).

Table 2 shows the association between insurance in the 6 months prior to interview and HIV-1 antibody status. HIV serostatus was not significantly associated with the presence of any type of health insurance.

Table 3 shows comparisons for the number of inpatient and outpatient medical visits between HIV seropositive and seronegative individuals. HIV seropositive individuals averaged significantly more inpatient admissions (0.21) in 6 months compared to HIV seronegative individuals (0.13). No significant difference was observed in the mean number of outpatient medical visits or inpatient drug or alcohol admissions by HIV-1 serostatus.

The effect of severity of illness on health service utilization was examined via two separate measures of severity, CD4 levels and presence of two or more HIV-related symptoms. Table 4 shows comparisons between use of health services, CD4 counts, and HIV-related symptoms. The CD4 counts of HIV positive individuals were stratified into three groups—<200, 200–500, and >500—and the mean number of visits was compared to that observed in a group of HIV seronegative IVDUs. HIV seropositive individuals with CD4 counts <200 and those with counts of 200–500 were significantly more likely to use inpatient services than a comparison group of HIV negative drug users ($P < .021$ and P .033, respectively). HIV positive individuals with CD4 levels <200 had higher numbers of outpatient visits than the comparison group of HIV seronegative IVDUs ($P < .079$). There was no significant difference in number of outpatient visits noted between HIV seropositive individuals with CD4 counts >200 compared to number of visits of HIV seronegative individuals.

The effect of symptoms on utilization of health services is also presented in Table 4. The mean number of outpatient visits of HIV seronegative individuals was compared to both HIV seropositive individuals with zero or one symptom and HIV positive individuals with two or more AIDS-related symptoms. Those with two or more symptoms were found to have significantly more inpatient admissions and outpatient visits than HIV seronegative individuals ($P < .001$ and $P < .003$, respectively).

Table 5 shows the relationship between insurance coverage and utilization of medical services in the prior 6 months. All individuals with health insurance, regardless of their HIV serostatus, had significantly more inpatient admissions, outpatient visits, or drug or alcohol treatment admissions than those without health insurance. We also noted a significant relationship (not shown) between utilization of emergency rooms and absence of health insurance ($P < .001$).

TABLE 2—Proportion of Intravenous Drug Users with Insurance and Health Service Use in Prior 6 Months Stratified by HIV-1 Serostatus

	HIV-1 Serostatus	
Characteristic	Positive (n = 610)	Negative (n = 1271)
Insurance status in past 6 months		
Medicaid	60.5	56.3
Private or other insurance	9.1	9.8
None	30.3	33.8

Overall $P = .227$

TABLE 3—Mean (±SD) Number of Medical Visits in Past 6 Months Stratified by HIV-1 Serostatus, Baltimore, Md

	Serostatus		
Variable	Positive n = 610	Negative n = 1271	P Value
Inpatient admissions	0.21 (0.55)	0.13 (0.48)	0.003
Outpatient visits	1.90 (5.05)	1.61 (4.07)	0.216
Inpatient drug or alcohol treatment	1.7 (5.04)	1.4 (4.65)	0.23

TABLE 4—Analysis of Variance for Medical Service Utilization among Intravenous Drug Users by HIV-1 Serostatus, CD4 Count, and Symptoms, Baltimore, Md

Variable	n	Mean Number Visits over past 6 Months	P value
Inpatient admission			
HIV−[a]	1271	0.14	—
HIV+, CD4 >500[b]	275	0.16	0.364
HIV+, CD4 200–500	207	0.21	0.033
HIV+, CD4 <200	34	0.56	0.021
HIV−[a]	1271	0.14	—
HIV+, 0–1 symptoms[c]	459	0.15	0.454
HIV+, ≥2 symptoms	92	0.57	0.001
Outpatient visit			
HIV−[a]	1271	1.61	—
HIV+, CD4 >500[b]	275	1.94	0.359
HIV+, CD4 200–500	207	1.74	0.612
HIV+, CD4 <200	34	3.61	0.079
HIV−[a]	1271	1.61	—
HIV+, 0–1 symptoms[c]	459	1.73	0.654
HIV+, ≥2 symptoms	92	3.41	0.003

[a] Reference group.
[b] 94 missing CD4 cells.
[c] 59 missing symptoms.

Multivariate Findings

Table 6 presents estimates from the two-stage instrumental variables probit model. (Estimates from the first stage of the model are presented in Appendix B. The rationale for the instrumental model specification is presented in Appendix C.) We expected to find a positive correlation between HIV positive serostatus, older age, symptoms, and insurance with both inpatient and outpatient health service utilization. Only one measure of severity—presence of two or more AIDS-related symptoms—was entered into the model. This measure was chosen instead of CD4 counts because data were available for all study participants and CD4 counts of <200 were strongly cor-

TABLE 5—Type of Health Service Utilization in Past 6 Months Stratified by HIV-1 Serostatus and Insurance

	Visit		No Visit		
Health Service	n	% with insurance	n	% with insurance	*P* value
Inpatient admission					
HIV positive	98	83.7	512	67.0	<.001
HIV negative	137	83.9	1134	64.0	<.001
Outpatient visit					
HIV positive	310	80.7	300	58.3	<.000
HIV negative	604	77.3	667	56.1	<.000
Inpatient detox					
HIV positive	73	87.7	532	66.9	<.001
HIV negative[a]	168	85.1	1099	63.2	<.000

[a]Missing 4.

TABLE 6—Final Probit Regression Estimates of Inpatient and Outpatient Utilization[b]

Variable	Inpatient Use[a]	Outpatient Use[a]
HIV-1 seropositive	0.127 (1.44)	0.044 (0.651)
Age 1 (25–34)	0.300* (1.62)	−0.198* (1.64)
Age 2 (35–44)	0.274 (1.03)	−0.101 (0.55)
Age 3 (≥45)	−0.011 (0.25)	0.06[illegible] (0.27)
Male gender	−0.318 (0.99)	0.070 (0.29)
Black race	0.204 (1.31)	0.091 (0.76)
Education (9–12)	0.090 (0.63)	0.809 (0.72)
Education (>12)	−0.061 (0.36)	0.189 (1.41)
Income ($5 000–$10 000)	−0.871 (0.69)	−0.163* (1.81)
Income ($10 000)	−0.208 (1.43)	−0.191* (1.81)
Symptoms of AIDS (≥2)	0.687** (4.77)	0.268** (2.19)
Insurance[a]	0.649* (1.63)	0.322 (1.12)
Intercept	−1.869** (8.61)	−0.178 (1.18)
Loglikelihood	−643.36	−1262.3

Note. Reference groups: age <25, female, non-Black, < 9 years.
[a]Endogenous variable.
[b]Asymptotic t statistics in parentheses.
*p < .10.
**p < .05.

related with the presence of two or more HIV-related symptoms. The first column of the model presents the results for the determinants of inpatient utilization, and the second column presents the results for outpatient utilization as a dichotomous variable. Although the results of the model for inpatient and outpatient are qualitatively similar for both models of utilization, there are some important differences. In terms of the variables of central concern, the results indicate that holding constant demographic factors and AIDS-related clinical markers, having insurance was marginally associated with the probability of inpatient care ($P <$ .06). However, insurance coverage has no significant impact on the use of outpatient care.

The variable representing HIV status was estimated to have a positive though not significant association with the probability of using either type of health care services. Although the coefficient estimate in the inpatient utilization model had a t statistic of 1.44, indicating a moderate amount of precision in the estimate, this was not significant by conventional standards. This suggests that holding constant demographic factors, insurance coverage, and symptoms, HIV serostatus did not have a significant independent impact on service utilization. Formal tests for interactions between HIV positive serostatus and the presence of health insurance showed no significant effects. As Table 6 shows, after adjusting for demographic variables, insurance, and HIV serostatus, the presence of two or more AIDS-related symptoms demonstrated the strongest association with the likelihood that an individual would get either inpatient or outpatient care.

Discussion

This study examined the relationship between health insurance, HIV-1 serostatus, and AIDS-related symptoms and the impact of these factors on the utilization of both outpatient and inpatient medical, drug, and alcohol treatment services. The presence of two or more AIDS-related symptoms had the strongest influence on the use of both inpatient and outpatient health care services. This is consistent with other research documenting the important role of symptoms and complaints in predicting service use.[18,19] In the multivariate analysis neither HIV-1 positive serostatus nor low CD4 levels in the absence of symptoms were associated with increased health services utilization. However, health insurance was significantly associated with the likelihood of using inpatient medical, drug, or alcohol treatment services. Even among individuals with low CD4 counts or AIDS-related symptoms, the absence of health insurance was associated with lower probabilities of receiving inpatient treatment. We did not find a significant relationship between the presence of health insurance and outpatient service utilization, which may be due in part to the availability of free care in East Baltimore. For example, the HIV clinics at certain local hospitals treat all individuals regardless of insurance status.

The next major finding is a negative one. Asymptomatic HIV seropositive subjects were estimated to be no more likely to use services than are HIV seronegative individuals. This is a particularly important problem because the greatest advances in treating HIV disease involve the postponement of manifestation of opportunistic infections that are the major causes of death in AIDS patients. Current medical practice recommends prophylactic treatment when CD4 counts drop below 500 mm^3, a level at which symptoms are not usually present. Failure to enroll HIV-positive individuals in health care prior to the appearance of symptoms suggests that preventive care, monitoring of CD4 counts, zidovudine prophylaxis, and prophylaxis for pneumocystis pneumonia are probably lacking and implies that individuals are not seeking care until they become very ill.

Programs to expand testing for HIV-1 in high-risk groups have been endorsed as an important element in promoting the early treatment of HIV-1 disease.[20] However, this study suggests that identification of HIV-1 positive status alone does not necessarily result in the initiation of preventive care. IVDUs often remain outside the health care system, using services on an emergent basis and frequenting emergency rooms rather than primary care clinics. Characteristics inherent in drug addiction (e.g., an overwhelming need to secure drugs daily) may make IVDUs particularly unable to conform to the requirements of many clinics for scheduled appointments and compliance with therapeutic plans. Efforts to encourage the early use of preventive services in this population will need to confront both the preferences and habits of the IV-drug-using population as well as institutional impediments to the use of services.[21,22]

Additionally, attention must be focused on making the drug-using community aware of the importance of early and continuous medical assessment and care. To date, education concerning HIV has focused on prevention of infection with implicit messages concerning the fatal consequences of HIV disease. Existing programs may have overlooked the need to provide information about the benefits of preventive treatment. Programs to expand medical services to drug treatment programs have become more common in Baltimore. Although this may increase knowledge and use of medical services to individuals participating in these drug treatment programs, our research suggests that the vast majority of drug users remain outside the drug treatment setting.

Conclusions based on this study may be limited by characteristics of the study population. This study reimbursed individuals for their participation; therefore, those with acute financial need may be overrepresented. Additionally, reports of insurance status as well as health services utilization were based on self-reports, the accuracy of which may be questionable. In spite of these potential limitations this study presents data on a cohort of drug users that are largely unaffiliated with a drug treatment program. Thus it may offer a more accurate picture of the use of services and the extent of care that many HIV seropositive drug users outside of treatment facilities are receiving. This study suggests that in spite of recommendations from the Centers for Disease Control for early treatment of HIV disease, many drug users still remain outside the health care system without benefit of preventive care. ☐

Acknowledgments

Portions of the preliminary data were presented at the 5th International Conference on AIDS in Montreal, Quebec, Canada (Abstract No. T.E.P.43).

This research was supported by the Agency for Health Policy and Research grant HS6411 and the National Institute on Drug Abuse grants DA04334 and DA05664.

We are grateful to Chuanfa Guo for his expert programming assistance and to the staff of the ALIVE study, without whom little of this work would have been possible. We also thank Dr. Joseph Margolick for his assistance with CD4 cell monitoring. Dr. Julia Hildalgo of the Maryland AIDS Administration gave invaluable assistance in verifying records.

References

1. National Institute on Drug Abuse. *AIDS and Intravenous Drug Abuse among Minorities.* Rockville, MD: US Department of Health and Human Services, 1989.
2. Bakeman R, Lumb JR, Smith DW. AIDS statistics and the risk for minorities. *AIDS Res Hum Retroviruses.* 1986;2:249–252.
3. Hahn RA, Onorato IM, Jones TS, Dougherty J. Prevalence of HIV infection among intravenous drug users in the United States. *JAMA.* 1989;261:2677–2684.
4. Centers for Disease Control. Leads from the MMWR: acquired immunodeficiency syndrome associated with intravenous-drug use—United States, 1988. *JAMA.* 1989;261:2314–2316.
5. Curran JW, Jaffe HW, Hardy AM, Morgan WM, Selik RM, Dondero TJ. Epidemiology of HIV infection and AIDS in the United States. *Science.* 1988; 239:610–616.
6. Green J, Arno PS. The 'medicaidization' of AIDS. *JAMA.* 1990;264:1261–1266.
7. Andrulis DP, Beers VS, Bentley JD, Gage LS. The provision and financing of medical care for AIDS patients in US public and private teaching hospitals. *JAMA.* 1987; 258:1343–1346.
8. Iglehart JK. Financing the struggle against AIDS. *N Engl J Med.* 1987;317:180–184.
9. Scitovsky AA. The economic impact of AIDS in the United States. *Health Aff.* 1988;Fall:7:32–45.
10. Centers for Disease Control. Guidelines for prophylaxis against *Pneumocystis carinii* pneumonia for persons infected with human immunodeficiency virus. *MMWR.* 1989;38(Supl 5):1–9.
11. Anthony JC, Vlahov D, Celentano DD, et al. Self-report interview data for a study of HIV-1 infection among intravenous drug users: description of methods and preliminary evidence on validity. *J Drug Issues.* In press.
12. Vlahov D, Anthony JC, Munoz A, et al. The ALIVE study: a longitudinal study of HIV-1 infection in IVDUs; description of methods. In: Amsel S, Genser SG, Hartsock PI, eds. *Longitudinal Studies of HIV Infection among IVDUs.* Washington, DC: NIDA Research Monograph; 1990; Senes No. 109, 1991.
13. Hoffman RA, Kung PC, Hansen WP, et al. Simple and rapid measurement of human T lymphocytes and their subclasses in peripheral blood. *Proc Natl Acad Sci.* 1980; 77:4914–4917.
14. Volberding PA, Lagakos SW, Koch MA, et al. Zidovudine in asymptomatic human immunodeficiency virus infection: a controlled trial in persons with fewer than 500 CD4-positive cells per cubic millimeter. *N Engl J Med.* 1990;322:941–949.
15. Brown BW, Hollander M. *Statistics: A Biomedical Introduction.* New York: John Wiley and Sons; 1977.
16. Maddala GS. *Limited-Dependent and Qualitative Variables in Econometrics.* Cambridge, England: Cambridge University Press; 1983.
17. Amemiya T. The estimation of a simultaneous equation probit model. *Int Econ Rev.* 1979;20:169–181.
18. Hulka BS, Wheat JR. Patterns of utilization: the patient perspective. *Med Care.* 1985;23:438–460.
19. Newhouse JP, Manning WG, Morris CN, et al. Some interim results from a controlled trial of cost sharing in health insurance. *N Engl J Med.* 1981;305:1501–1507.
20. Rhame FS, Maki DG. The case for wider use of testing for HIV infection. *N Engl J Med.* 1989;320:1248–1254.
21. Des Jarlais DC, Friedman SR, Hopkins W. Risk reduction for the acquired immunodeficiency syndrome among intravenous drug users. *Ann Intern Med.* 1985; 103:755–759.
22. Dans PE, Matriciccinai RM, Otter SE, Reuland DS. Intravenous drug abuse and one academic health center. *JAMA.* 1990; 263:3173–3176.

APPENDIX A—HIV-Related Symptoms

Tired all the time for 2 or more weeks
Shortness of breath for 2 or more weeks
Diarrhea for 2 or more weeks
Unintentional weight loss of 10 or more pounds
White patches in the mouth for 2 or more weeks

APPENDIX B—First Stage Probit Regression Estimates for Insurance

Variable	Insurance[a]
HIV positive	0.73 (1.05)
Age 1 (25–34)	0.154 (1.32)
Age 2 (35–44)	0.495[a] (4.07)
Age 3 (≥45)	0.914[a] (5.35)
Male	0.722[a] (7.99)
Black	−0.162 (1.41)
Education 1 (9–12)	−0.058 (0.49)
Education 2 (>12)	−0.032 (0.22)
Income 1 ($5,000–$10,000)	−0.061[a] (0.69)
Income 2 (>$10,000)	0.226 (2.20)
Children	0.097 (1.41)
Employed	−0.240[a] (3.13)
AIDS symptoms	0.245[a] (2.45)
Intercept	0.041 (0.25)
Loglikelihood	−1067.3

Asymptotic *t* statistics in parentheses.
[a] $p \leq .05$.

APPENDIX C—Rationale for Instrumental Variables Model Specification

Within the model specification it is important to include at least some independent variables that are not included as part of the utilization model. While this is difficult to accomplish on a priori grounds, we choose two variables that relate to the price of insurance. This is consistent with sequential budgeting on the part of consumers. Under such arrangements consumers first make a choice regarding the purchase of insurance and then subsequently choose to buy other goods based on the remaining income. Under such a model, variables affecting the price of insurance will have little effect on utilization. The two variables that differ between the two models are whether or not individuals have children and whether or not they are employed. We posit that these variables have a strong impact on the likelihood of having insurance and little effect on utilization once income, demographic features, and health status are taken into account.

Risk Factors for Shooting Gallery Use and Cessation among Intravenous Drug Users

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ABSTRACT

Background. Shooting galleries, locations where intravenous drug users (IVDUs) can rent or borrow needles and syringes, are a high-risk environment for HIV-1 transmission. This study investigates risk factors for lifetime attendance at shooting galleries and differentiates characteristics of those who continue to frequent shooting galleries and those who have stopped.

Methods. We interviewed 2615 active IVDUs in Baltimore in 1988 and 1989 and determined patterns of IV drug use, sociodemographics, and HIV-1 serostatus as related to persistence vs cessation of shooting gallery use.

Results. Over half (52%) of active IVDUs reported ever using a shooting gallery, with 33% reporting use within the prior 3 months. In multivariate analysis, lifetime shooting gallery use was associated with male gender, homosexuality/bisexuality, low socioeconomic status, Black race, and heavier drug involvement. Persistent shooting gallery users were more frequently male, homosexual/bisexual, homeless, less educated, and started IV drug use more recently compared with those who ceased going to shooting galleries.

Conclusions. Shooting gallery attendance may be pragmatic from a sociological and economic perspective, but it carries with it a heightened risk of acquiring HIV-1 infection. (*Am J Public Health*. 1991;81:1291–1295)

Introduction

Behaviors that increase the risk of infection with human immunodeficiency virus, type 1 (HIV-1) among intravenous drug users (IVDUs) are primarily those involving transfer of blood through shared injection equipment.[1-5] Use of shared equipment at shooting galleries is particularly high risk for acquiring HIV-1 infection.[6-8] Shooting galleries are locations, generally clandestine, where IVDUs can rent, borrow, or purchase needles and syringes; frequently the injection equipment is used repeatedly and may contain contaminated blood. Repeated rental of syringes results in a sequential anonymous sharing of potentially contaminated equipment. Such sharing facilitates the transmission of blood-borne pathogens.[1]

Although needle sharing has been empirically demonstrated to be one of the principal AIDS risk behaviors among IVDUs,[9,10] reports on the characteristics and behaviors of IVDUs who use shooting galleries are sparse. Ethnographic reports suggest that IVDUs go to shooting galleries for several reasons, including (1) the illegality of possession of drug injection equipment and consequent fears of arrest; (2) the scarcity of syringes, leading to an economic imperative to share injection equipment; and (3) the social context of intravenous drug use.[11,12] In this report, we used social survey methods on a large sample of currently active IVDUs to complement the findings from the published ethnographic reports to examine reasons for shooting gallery use and to identify reasons for cessation vs persistence in gallery use.

Methods

Sample

Between February 1988 and March 1989, IVDUs in Baltimore, Md, were recruited into a longitudinal investigation of the natural history of HIV-1 infection [the AIDS Links to the Intravenous Experience (ALIVE) Study]. Recruitment relied predominately on word-of-mouth by community outreach efforts (85%); referrals from drug abuse treatment programs, sexually transmitted disease clinics, local emergency departments, university hospital HIV-1/AIDS clinics, and shelters for the homeless also assisted with cohort recruitment. In order to be eligible for enrollment, IVDUs had to be age 18 or older and have self-reported illicit drug injection in the prior 10 years. The rationale and details of the sampling and data collection plan have been described elsewhere.[13]

A total of 2921 participants were enrolled in the ALIVE study and completed baseline interviews and venipuncture. In this analysis, we restricted the sample to the 2615 IVDUs who reported injecting illicit drugs within 6 months prior to interview.

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This paper was submitted to the journal July 13, 1990, and accepted with revisions March 7, 1991.

Data Collection Procedures

After providing voluntary informed consent, participating IVDUs were assigned a unique and confidential identifier and agreed to venipuncture for serum for HIV-1 antibody assays. Next, participants were questioned by a trained interviewer in a private room concerning demographic information, medical history, drug injection practices since 1977 on an annual basis, 10-year history of sexual activity, knowledge and attitudes about HIV-1 infection and AIDS, and use of medical care. A detailed description of the data-gathering procedures appears elsewhere[14]; a brief overview is presented here.

The self-report demographic data analyzed here include age, gender and sexual orientation, race/ethnicity, education, current employment, receipt of public assistance, and occurrence of homelessness within the prior 10 years. Participants were asked to recall their drug use behavior in the 6 months prior to their last injection and also in the first 3 months of use. Drug injection frequency reported for the prior year was categorized as once or more a day or as less frequently. Other drug involvement factors analyzed included self-reports of treatment for drug abuse, being arrested and incarcerated within the prior 10 years, and duration of drug use as indicated by the year when drugs were first injected. Although there was a battery of questions on history of sexual practices, we restricted the analysis here to one indicator: whether any sexual partner was an IVDU.

We inquired as to the use of shooting galleries on an annual basis (thereby determining never and ever use) beginning in 1977 or year of first drug use. Recent shooting gallery attendance was ascertained as follows: "During the 6 months before the last time you injected drugs, how many times did you go to a shooting gallery and use works borrowed, rented, or bought there?" For the analyses here we aggregated the responses into three categories: never went to a shooting gallery, former shooting gallery user but no use in the prior 6 months, and current user of shooting galleries (i.e., attendance within the prior 6 months).

Analysis

To examine the associations of sociodemographic and drug use characteristics with attendance at shooting galleries, we cross-classified current, former, and never users of shooting galleries with the predictor characteristics, using χ^2 statistics as an aid to interpretation. The strength of associations between these characteristics and practices and shooting gallery attendance was estimated by the odds ratio (OR), using the group of IVDUs who reported never going to a shooting gallery as the reference category. Logistic regression was used to examine the simultaneous association of these factors to lifetime shooting gallery use (comparing ever vs never), as well as to identify factors associated with persistence of this risk behavior (comparing current and former users). We used 95% confidence intervals (CIs) as an aid to statistical interpretation of results.

Results

Of the 2615 IVDUs still injecting drugs enrolled in the ALIVE study, 32.9% (n=861) reported attendance at a shooting gallery within the prior 6 months, and 19.4% (n=507) reported having gone to a shooting gallery in the past but not within the prior 6 months. Slightly less than one half (47.7%; n=1247) of the respondents denied ever having injected drugs in a shooting gallery.

The relationships between the demographic and drug use characteristics and lifetime shooting gallery attendance are presented in Table 1. Those IVDUs who have ever used shooting galleries were more likely to be male, homosexual/bisexual, slightly older, Black, and unemployed, to have received public assistance, and to report being homeless sometime in the prior decade, as compared to IVDUs who reported never injecting drugs in a shooting gallery. A review of drug involvement factors shows shooting gallery attenders were more likely to report an arrest or incarceration history, to have been in drug treatment as compared to never users, and to have initiated injection behavior earlier in their lives. With respect to current drug use, shooting gallery users were more likely to be daily drug users and to report sexual partners who were also IVDUs. Table 2 compares the characteristics of current shooting gallery users and IVDUs who reported stopping shooting gallery use. Current shooting gallery attenders were, again, male, homosexual/bisexual, younger, less educated, more often homeless in the prior decade, and had a history of injection behavior showing more recent initiation into this culture. No other drug involvement factor differentiated those persisting in shooting gallery use and those who ceased this activity.

Table 3 presents the results of two multiple logistic regression analyses: (1) ever vs never use of shooting galleries and (2) current vs former shooting gallery users. Age was deleted so that year of first IV drug use might be included in the equations. Using multiple regression to characterize those IVDUs who reported ever going to a shooting gallery compared with those with no self-report history of gallery attendance, we found that ever attenders were more likely to be male, homosexual/bisexual, Black, and homeless, to report arrests, drug treatment, and sex partners who were IVDUs, and to have a history of daily drug injection and of starting drug injection earlier, all other variables held constant. Compared to females, males, especially homosexuals or bisexuals, more commonly visited shooting galleries. The comparison of characteristics differentiating individuals who persist in injecting illicit drugs in shooting galleries and those who have stopped showed fewer distinguishing factors once other variables were held constant using multiple regression: recent gallery attenders had somewhat less education, more frequently reported homelessness, and more recently initiated IV drug use. The high OR for male homosexuals or bisexuals who persist in using shooting galleries is of concern because they may serve as a bridge for HIV-1 transmission from gay males who do not inject to heterosexual IVDUs.

Discussion

These data indicate that shooting gallery use is associated with lower socioeconomic status, greater criminal involvement, and indicators of heavier drug use—characteristics congruent with prior ethnographic reports of street drug users.[15–17] Our findings suggest that the use of shooting galleries is in part a pragmatic response to the contemporary context of illicit drug use, namely, that IVDUs seek access to needles for injecting drugs that are illegal to possess.[18] Shooting galleries provide needles in relatively clandestine locations, also reducing the threat of arrest for drug use and drug and drug paraphernalia possession.[19,20] However, the lack of financial resources associated with shooting gallery use fails to differentiate IVDUs who have stopped using shooting galleries from those that persist in this behavior, with the exception of greater reports of homelessness among recent shooting gallery users. Homeless individuals are more likely to frequent shooting galleries and to persist in this behavior, thereby providing some limited evidence supporting an hypothesis of economic necessity. The traditional view that needle sharing reflects a culturally entrenched ritual resistant to change[21] has been expanded to acknowledge a pragmatic response of IVDUs to the restricted availability of needles and syringes.[22] Lessons learned from studies of needle sharing offer some insights into the complexity of this social behavior but appear insufficient to fully explain its continued existence.

While going to shooting galleries may be pragmatic from a sociolegal and economic perspective, using needles obtained from shooting galleries is associated with increased risk of acquiring HIV-1 infection. The extent of exposure to HIV-1 in shooting galleries was unknown until recently. Chitwood et al.[23] obtained 148 needles and syringes from three shooting galleries in Miami, of which 10.1% tested positive for HIV-1 antibody. This relatively high rate of exposure found in shooting galleries may reflect frequent reuse of equipment as well as sharing equipment more frequently with strangers or acquaintances whose HIV-1 antibody status may be unknown (as compared to sharing needles among groups of close friends).[24,25] In our study, the odds of having antibody to HIV-1 among those who have ever gone to a shooting gallery were nearly twice the odds for those who had reported never attending a gallery.[8] Vigorous prosecution of drug paraphernalia offenders may act to facilitate transmission of HIV-1 infection rather than reduce its spread.

One public health approach to the problem of HIV-1 transmission in shooting galleries is to offer sterile needles through needle exchange programs.[26-28] This approach generally has been considered incompatible with current policies on drug use in most US jurisdictions. A recent report[7] discusses public health preventive interventions to reduce the risk of HIV-1 among users of shooting galleries in the context of the current legal climate—educating proprietors of shooting galleries to educate their clients and to improve needle sterilization practices. This approach requires the active participation of shooting gallery operators in creating "safer" galleries; additional studies will be required to determine the extent to which such interventions are feasible and effective. Ultimately, a goal for reducing the transmission of HIV-1 infection among IVDUs would involve the cessation of shooting gallery use, rather than attempts to create "safer" galleries. Study results presented here suggest that although a large proportion (37%) of IVDUs reporting ever going to shooting galleries have recently stopped, the majority persist in this activity. Data on AIDS knowledge and attitudes posed to these participants demonstrated no trends differentiating these three groups of gallery users.

TABLE 1—Percentage Distributions and Odds Ratio (OR) Estimates of Sociodemographic and Drug Use Practices on Lifetime Shooting Gallery Attendance

	Shooting Gallery Attendance			
	Never (n = 1247)	Ever (n = 1368)	OR	95% CI[a]
Gender/Orientation				
Female	23.7	14.1	1.0	
Male/Heterosexual	70.0	76.4	1.8	1.5–2.2
Male/Bisexual	6.3	9.5	2.5	1.8–3.5
Age (years)				
18–24	8.6	5.7	1.0	
25–29	22.9	16.6	1.1	0.8–1.6
30–34	29.0	29.6	1.5	1.1–2.2
35–39	21.1	26.6	1.9	1.3–2.7
≥40	18.4	21.5	1.5	1.2–2.5
Race				
White	12.9	6.7	1.0	
Black	87.1	93.3	2.0	1.6–2.7
Education (years)				
≤12	55.8	56.8	1.0	
>12	44.2	43.2	1.0	0.9–1.2
Currently employed				
Yes	23.7	19.2	1.0	
No	76.3	80.8	1.3	1.1–1.6
Public assistance				
No	35.0	25.7	1.0	
Yes	65.0	74.3	1.6	1.3–1.8
Homeless[b]				
No	57.3	45.6	1.0	
Yes	42.7	54.4	1.6	1.4–1.9
Arrested[b]				
No	30.2	17.0	1.0	
Yes	69.8	83.0	2.1	1.8–2.6
Incarcerated[b]				
No	40.3	27.3	1.0	
Yes	59.7	72.7	1.8	1.5–2.1
Drug treatment (ever)				
No	59.1	37.0	1.0	
Yes	40.9	63.0	2.4	2.1–2.8
Year initiated IV drug use				
1988	7.3	1.5	1.0	
1986–1987	13.0	6.0	2.3	1.3–4.0
1984–1985	8.5	5.0	3.0	1.7–5.2
1980–1983	15.9	12.1	3.8	2.3–6.5
Before 1980	55.3	75.4	6.8	4.2–11.1
Frequency of drug injection				
< Daily	69.2	51.3	1.0	
Daily	30.8	48.7	2.1	1.8–2.5
IVDU[c] sex partner				
No	35.1	17.5	1.0	
Yes	64.9	82.5	2.6	2.1–3.1

[a] CI = confidence interval.
[b] During the previous 10 years.
[c] IVDU = intravenous drug user.

A focus on risk reduction among homosexual or bisexual males who inject drugs in shooting galleries seems to be of paramount importance. Although patterns of shooting gallery use fluctuate over time and our classification into three distinct groups based on recent history may be arbitrary, no distinct trends in use were detected as related to the risk factors assessed. Although other studies report behavior change among IVDUs in response to the HIV-1 epidemic,[29] these

TABLE 2—Percentage Distributions and Odds Ratio (OR) Estimates of Sociodemographic and Drug Use Practices on Current vs Former Shooting Gallery Attendance

	Shooting Gallery Attendance			
	Current (n = 861)	Former (n = 507)	OR	95% CI[a]
Gender/orientation				
Female	12.1	17.7	1.0	
Male/Heterosexual	76.0	77.2	1.4	1.1–2.0
Male/Bisexual	11.9	5.1	3.4	2.0–5.7
Age, y				
18–24	7.3	3.0	1.0	
25–29	17.4	15.2	0.5	0.3–0.9
30–34	29.3	30.2	0.4	0.2–0.7
35–39	24.3	30.6	0.3	0.2–0.6
≥40	21.7	21.1	0.4	0.2–0.8
Race				
White	6.4	7.3	1.0	
Black	93.6	92.7	1.1	0.8–1.6
Education, y				
≤12	59.4	52.5	1.0	
>12	40.6	47.5	1.3	1.1–1.7
Currently employed				
Yes	18.9	19.5	1.0	
No	81.1	80.5	1.0	0.8–1.4
Public assistance				
No	26.0	25.3	1.0	
Yes	74.0	74.7	1.0	0.8–1.2
Homeless[b]				
No	41.7	52.3	1.0	
Yes	58.3	47.7	1.5	1.2–1.9
Arrested[b]				
No	17.1	16.8	1.0	
Yes	82.9	83.2	1.0	0.7–1.3
Incarcerated[b]				
No	25.9	29.6	1.0	
Yes	74.1	70.4	1.2	0.9–1.5
Drug treatment (ever)				
No	36.5	37.9	1.0	
Yes	63.5	62.1	0.9	0.7–1.2
Year initiated IV drug use				
1988	2.2	0.2	1.0	
1986–1987	7.9	2.8	0.3	0.1–2.1
1984–1985	5.0	5.1	0.1	0.1–0.7
1980–1983	11.4	13.4	0.1	0.1–0.6
Before 1980	73.5	78.5	0.1	0.1–0.6
Frequency of drug injection				
< Daily	50.3	53.1	1.0	
Daily	49.7	46.9	1.1	0.9–1.4
IVDU[c] sex partner				
No	16.0	19.9	1.0	
Yes	84.0	80.1	1.3	0.9–1.7

[a] CI = confidence interval.
[b] During previous 10 years.
[c] IVDU = intravenous drug user.

studies note that a substantial proportion of IVDUs persist in risk-increasing behaviors.[30] Further, studies have shown that although some IVDUs who engage in needle sharing practices are knowledgeable about modes of transmission,[31,32] they do not perceive themselves to be at high risk for infection.[33] These data are consistent with previous reports[29,30] indicating that a large proportion of IVDUs continue to engage in high risk behaviors, despite their knowledge about HIV-1.

The characteristics of those who continue to use shooting galleries suggest that we need to target IVDUs earlier in their careers, before this behavior becomes firmly entrenched, and to focus additional interventions on an especially difficult-to-access subset of IVDUs—those who are less educated and impoverished. Paradoxically, those who persist in using shooting galleries are also likely to report a history of drug treatment, a characteristic shown to lead to reduced IV drug use[34] although relapse is frequent.[35] However, those who continue going to shooting galleries are not necessarily more frequent drug users than those who have stopped, suggesting that overall drug involvement is not a discriminating characteristic. HIV-1 prevention efforts must strive for greater access to IVDUs who use shooting galleries in order to appropriately target messages on cessation of shooting gallery use. Prevention efforts should also consider the approach suggested by Chitwood et al.[23] and articulated by Des Jarlais and Friedman[7] of making galleries safer for those who use them. ☐

Acknowledgments

Supported in part by Public Health Service grants DA04334 and DA05911, awarded by the National Institute on Drug Abuse.

This paper was presented in preliminary form at the Sixth International Conference on AIDS, San Francisco, Calif, June 23, 1990.

References

1. Friedland GH, Klein RS. Transmission of the human immunodeficiency virus. *N Engl J Med.* 1987;31:1125–1135.
2. Des Jarlais DC, Friedman SR, Stoneburner RL. HIV infection and intravenous drug use: critical issues in transmission dynamics, infection outcomes, and prevention. *Rev Infect Dis.* 1988;10:151–158.
3. Chaisson RE, Moss AR, Onishi R, Osmond D, Carlson JR. Human immunodeficiency virus infection in heterosexual intravenous drug users in San Francisco. *Am J Public Health.* 1987;77:169–172.
4. Shoenbaum EE, Hartel D, Selwyn PA, et al. Risk factors for HIV-1 infection in intravenous drug abusers. *N Engl J Med.* 1989;321:874–879.
5. Dolan MP, Black JL, Deford HA, Skinner JR, Robinowitz R. Characteristics of drug abusers that discriminate needle sharers. *Public Health Rep.* 1987;102:395–398.
6. Marmor M, Des Jarlais DC, Cohen H, et al. Risk factors for infection with human immunodeficiency virus among intravenous drug abusers in New York City. *AIDS.* 1987;1:39–44.
7. Des Jarlais DC, Friedman SR. Shooting galleries and AIDS: infection probabilities and 'tough' policies. *Am J Public Health.* 1990;80:142–144. Editorial.
8. Vlahov D, Munoz A, Anthony JC, Cohn S, Celentano DD, Nelson KE. Association of drug injection patterns with antibody to human immunodeficiency virus, type 1 among IVDUs in Baltimore. *Am J Epidemiol.* 1990;132:847–856.
9. Chaisson RE, Bacchetti P, Osmond D, Brodie B, Sande MA, Moss AR. Cocaine use and HIV infection in IVDUs in San Francisco. *JAMA.* 1989;261:561–565.

TABLE 3—Multiple Logistic Regression Analysis Results on Lifetime and Recent Shooting Gallery Attendance

	Ever vs Never		Current vs Former	
	OR	95% CI	OR	95% CI
Gender:				
Male heterosexual	2.1	1.7–2.7	1.8	1.3–2.5
Male homosexual/bisexual	2.5	1.7–3.6	4.1	2.4–7.2
Race: Black	2.6	1.9–3.5	1.3	0.8–2.1
Education: ≤12 years	1.1	0.9–1.3	1.3	1.1–1.7
Currently employed: No	1.0	0.9–1.3	1.0	0.7–1.4
Public assistance: Yes	1.2	1.0–1.5	0.9	0.7–1.3
Homelessness: Yes	1.4	1.1–1.7	1.6	1.2–2.0
Arrested: Yes	1.4	1.1–1.8	0.7	0.5–1.1
Incarcerated: Yes	1.2	0.9–1.4	1.2	0.9–1.6
Drug treatment: Yes	1.9	1.6–2.3	1.3	1.0–1.6
Daily injection: Yes	1.8	1.5–2.2	1.3	1.0–1.6
Year started IV drug use	1.3	1.2–1.4	0.7	0.6–0.8
Sex partner IVDU: Yes	2.3	1.9–2.8	1.3	0.9–1.7
Model χ^2	463.4, $P<.0001$		77.3, $P<.0001$	

Note. OR=odds ratio; CI=confidence interval; IVDU=intravenous drug user.

10. Turner CF, Miller HG, Moses LE, eds. *AIDS, Sexual Behavior, and Intravenous Drug Use.* Washington, DC: National Academy Press; 1989.
11. Preble E, Casey JJ. Taking care of business—the heroin user's life on the street. *Int J Addict.* 1969;4:1–24.
12. Feldman HW. Ideological supports to becoming and remaining a heroin addict. *J Health Soc Behav.* 1968;9:131–139.
13. Vlahov D, Anthony JC, Munoz A, et al. The ALIVE study: a longitudinal study of HIV-1 infection in IVDUs; description of methods. In: Hartsock PI, Genser SG, eds. *Longitudinal Studies of HIV Infection among IVDUs.* National Institute on Drug Abuse Research Monograph 1991 (in press).
14. Anthony JC, Vlahov D, Celentano DD, et al. Self-report interview data for a study of HIV-1 infection among intravenous drug users: description of methods and preliminary evidence on validity. *J Drug Issues.* In press.
15. Hanson B, Beschner G, Watters JM, Boville E. *Life with Heroin: Voices from the Inner City.* Lexington, Mass: D.C. Heath; 1985.
16. Fiddle S. *Portraits from a Shooting Gallery.* New York, NY: Harper & Row; 1967.
17. Watters JM. Observations on the importance of social context in HIV transmission among IVDUs. *J Drug Issues.* 1989;19:9–26.
18. Murphy S. Intravenous drug use and AIDS: notes on the social economy of needle sharing. *Contemp Drug Prob.* 1987; 14:373–395.
19. Howard J, Borges P. Needle sharing in the Haight: some social and psychological functions. *J Health Soc Behav.* 1970;11: 220–230.
20. Ghodse AH, Treganza G, Li M. Effect of fear on sharing injection equipment among drug abusers. *Br J Med.* 1987;295:698–699.
21. Feldman HW, Biernacki P. The ethnography of needle sharing among IVDUs and implications for public policies and intervention strategies. In: Battjes RJ, Pickens RW, eds. *Needle Sharing among IVDUs: National and International Perspectives.* Washington, DC: National Institute on Drug Abuse; 1988. NIDA Research Monograph No. 80:28–39.
22. Des Jarlais DC, Friedman SR, Strug D. AIDS and needle sharing within the IV-drug use subculture. In: Feldman DA, Johnson TM, eds. *The Social Dimensions of AIDS: Method and Theory.* New York, NY: Praeger; 1986:111–125.
23. Chitwood DD, McCoy CB, Inciardi JA, *et al.* HIV seropositivity of needles from shooting galleries in South Florida. *Am J Public Health.* 1990;80:150–152.
24. Friedman SR, Des Jarlais DC, Sotheran JL. AIDS and self-organization among IV-DUs. *Int J Addict.* 1987;22:201–219.
25. Battjes RJ, Pickens RW, eds. *Needle Sharing among Intravenous Drug Users: National and International Perspectives.* Washington, DC: National Institute on Drug Abuse; 1988. NIDA Research Monograph No. 80.
26. Stimson GV. Syringe-exchange programmes for injecting drug users. *AIDS.* 1989;3:253–260.
27. Hart GJ, Carvell ALM, Woodward N, Johnson AM, Williams P, Parry JV. Evaluation of needle exchange in central London: behavior change and anti-HIV status over one year. *AIDS.* 1989;3:261–265.
28. Donoghoe MC, Stimson GV, Dolan K, Alldritt L. Changes in HIV risk behaviour in clients of syringe-exchange schemes in England and Scotland. *AIDS.* 1989;3:267–272.
29. Vlahov D, Anthony JC, Celentano DD, Solomon L, Chowdhury N. Trends of risk reduction among initiates into intravenous drug use 1982–1987. *Am J Drug Alcohol Abuse.* 1990;17:39–48.
30. Magura S, Grossman JI, Lipton DS, et al. Determinants of needle sharing among intravenous drug users. *Am J Public Health.* 1989;79:459–462.
31. Friedman SR, Des Jarlais DC, Sotheran JL. AIDS health education for IVDUs. *Health Educ Q.* 1986;13:383–393.
32. Ginzburg HM, French J, Jackson J, Hartsock PI, MacDonald MG, Weiss SH. Health education and knowledge assessment of HTLV-III diseases among IVDUs. *Health Educ Q.* 1986;13:373–382.
33. Celentano DD, Vlahov D, Menon AS, Polk BF. HIV knowledge and attitudes among IVDUs—comparisons to the US population and by drug use behaviors. *J Drug Issues.* 1991;21:647–661.
34. Abdul-Quader AS, Friedman SR, Des Jarlais D, Marmor MM, Maslansky R, Bartelme S. Methadone maintenance and behavior by intravenous drug users that can transmit HIV. *Contemp Drug Prob.* 1987;11:425–434.
35. Strug DL, Hunt DE, Goldsmith DS, Lipton DS, Prunt B. Patterns of cocaine use among methadone clients. *Int J Addict.* 1985;20:1163–1175.

Comparing the Predictive Accuracy of Health Risk Appraisal: The Centers for Disease Control Versus Carter Center Program

ABSTRACT

Background. From 1986 to 1987 the Carter Center of Emory University joined with the Centers for Disease Control (CDC) to develop a new, probability-based, adult health risk appraisal (HRA) instrument for the public domain. This new instrument is compared with the CDC HRA version to determine which is a more accurate predictor of mortality.

Methods. We compared predicted mortality risks from the CDC HRA and the Carter Center HRA with the observed mortality among 3135 smokers and never-smoking persons, aged 25 to 60, followed from 1959 to 1979 as part of the Tecumseh Community Health Study.

Results. When individuals were classified according to the difference between their actual age and risk age, for the CDC HRA, there was a progressively increasing risk of 10-year mortality as the difference increased. The Carter Center HRA did not show this trend. An analysis using relative operating characteristic curves showed that the mortality risk predictions for both programs were very similar for men and women. However, differences between actual age and risk age for the two programs were not similar for men or women, particularly older men. Therefore, actual age minus risk age for the CDC program was a more accurate predictor of 10-year mortality than was this difference for the Carter Center program.

Conclusions. The results from both types of analyses suggest that the validity of risk ages obtained from the Carter Center version may not be sufficient to justify updating programs for those currently using the CDC instrument. (*Am J Public Health.* 1991;81:1296–1301)

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Introduction

The health risk appraisal (HRA) has become a popular approach to help people identify the risks associated with personal characteristics (biological, life-style, family history).[1] Risk factors identified from various epidemiologic studies are combined with mortality statistics in order to assess an individual's risk of mortality within the next 10 years. An individual's risk is usually expressed as risk age (or appraised age), which enables one to compare one's own modifiable risk with that of a cohort. Thus the HRA could be a reasonably efficient method for transmitting this mortality risk information to individuals and stimulating them to change in terms of attitudes, beliefs, and life-styles, if so indicated.[2,3]

Although there are numerous variations of the HRA instrument, the majority are adapted from techniques developed by Robbins and Hall.[4] The accuracy of the mortality predictions from this methodology has been questioned repeatedly.[1–3,5–9] For example, Smith et al.[10] report that this method systematically overestimates the probability of mortality from coronary heart disease when the predictions are correlated with those from the Framingham Heart Study and the Risk Factor Update Project. Foxman and Edington assessed the accuracy of the Centers for Disease Control (CDC) HRA by comparing HRA-predicted risks of mortality with deaths that actually occurred among 3135 persons followed from 1959 to 1979 as part of the Tecumseh Community Health Study.[7] They found that the differences between actual age and CDC HRA risk age correctly classified individuals into high- and low-risk groups.

In an effort to update the scientific base of the HRA instrument, the Carter Center of Emory University joined with the CDC from 1986 to 1987 to develop a new, probability-based, adult health risk appraisal instrument for the public domain. The result of this project is "Healthier People," the Carter Center of Emory University Health Risk Appraisal Program.[11] The Carter Center HRA incorporates mortality tables from 1980 to 1982; integrates recommendations from the Breslow Risk Factor Update Project; uses regression equations from heart attack and stroke estimates developed by the Framingham Study; employs new models for cancer estimates developed from National Cancer Institute data; corrects height-weight recommendations; includes high-density lipoprotein as a risk factor; eliminates race as a predictor variable, so that mortality estimates are based only on age and sex; replaces the credit-debit method with multivariate statistical techniques; adjusts reference mortality projections for causes of death where risks are quantified by nonmodifiable precursors, so that individuals are not penalized if they have high levels of fixed risk, e.g., family history of breast cancer, diabetes, etc.; and calculates an individual's risk from 19, rather than 11, quantifiable causes of

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death. The Carter Center promises continuous updating of software as new scientific findings are determined and evaluated and technical enhancements developed.[11,12]

We have compared this new HRA instrument (version 3.1, released in late 1988) with the CDC HRA version that was used in our previous study[7] to see if this new version more accurately predicts mortality. This comparison has important implications for those using the HRA instrument for scientific and research purposes as well as for those using it for health education or intervention.

Methods

As in our previous study,[7] data available from the Tecumseh Community Health Study were used as input to the HRA programs. In order to compare the two instruments, we calculated the average HRA-predicted 10-year mortality risk (predicted risks) and risk age for both programs.

Risk age is defined for a given individual as the expected age of someone in the reference population of the same age, sex, and other fixed characteristics for whom the average 10-year mortality risk is the same as the HRA-predicted 10-year mortality risk. Thus, for the CDC HRA method a risk age of 50 for a 40-year-old White male means that the 10-year mortality risk predicted for that person is equal to the life-table estimate of the 10-year mortality risk for a 50-year-old White male in the 1975 to 1977 US population (the reference population for the CDC HRA). Hence this 40-year-old person is in poorer health than someone of the same age, sex, and race in the reference population.

The Carter Center HRA compares the predicted risk to the cohort average risk mortality, i.e., the population average risk for a person of the same age and sex adjusted for the presence of nonmodifiable risk factors.[11] Thus an individual with a family history of breast cancer would be compared with a reference population different from that of an individual without a family history of breast cancer. This reference mortality adjustment is made only for causes of death where risks are quantified by nonmodifiable precursors (US population projections are used as the reference mortality for all other causes of death).[12] A detailed comparison between the CDC HRA and the Carter Center HRA calculation of an individual's predicted risk of mortality and risk age is outlined in Appendix A.

Data Instruments

The Carter Center HRA (version 3.1) computes health risks based on a 45-item questionnaire[11]; the CDC HRA uses a 37-item questionnaire.[13] Twenty-nine of the 45 questions for the Carter Center HRA and 31 of the 37 questions for the CDC HRA are used directly for computation of risk (predicted risks and risk age, Appendix B). These questions were selected by the program's developers for their presumed causal relationship rather than mere statistical association. When items are not answered, both programs assign population norms.

Study Population

The Tecumseh Community Health Study began in 1959 as a comprehensive prospective investigation of health and disease in a community.[14] The principal aim was to identify causes and precursors of coronary heart disease, hypertension, chronic respiratory disease, diabetes mellitus, and other chronic diseases. Mortality ascertainments were completed successfully on more than 99% of previously examined subjects on three occasions, the most recent being 1978 to 1979.

In the 20-year period following the first cycle of examinations, 1059 (12.3%) of the 8641 participants died. Since the HRA is considered most accurate in the 25- to 60-year age range, we limited our analyses to the 3762 persons aged 25 to 60 in 1959 to 1960. Time since stopped smoking was not available for the 564 former smokers but is required by the CDC HRA; excluding former smokers from the current study restricted our sample to 3198; vital status after 10 years was available for 3166, and 3135 had sufficient information to calculate HRA. Thus a total of 3135 never-smoking or currently smoking individuals aged 25 to 60 in 1959 were included in this analysis.

Of the 29 variables used by the Carter Center HRA in predicting risk age, 13 were collected during the 1959 to 1960 Tecumseh survey, and 14 of the 31 variables used by the CDC HRA were available (Appendix B). Some recoding of the Tecumseh survey responses (mainly collapsing of categories to correspond to the precoded responses of both HRAs) was necessary for six of the variables. Although the wording was somewhat different in the two HRA instruments, the meanings were judged to be the same.

Data Analysis

Using both programs for each individual from data gathered during the 1959 to 1960 Tecumseh Community Survey, we compared the predictions of the CDC HRA with the Carter Center HRA, calculating the average HRA-predicted 10-year mortality risk from all causes (predicted risks) and risk age. The difference between each individual's age at the baseline survey and risk age (age difference) was compared by gender and age groups. To determine if risk age was a good predictor of observed mortality, we examined the proportion dying at 10 years for both men and women by the difference between actual age and risk age. The average HRA-predicted mortality risk, observed mortality, and age-adjusted mortality risk were calculated for each category of this difference between actual age and risk age. Since variations in age, sex, and race explain much of the variability in death rates, examining the gender, age, and race-specific differences between actual age and risk age represents the contribution of nondemographic inputs to the HRA.[15]

In order to assess which instrument more accurately predicted mortality, we performed relative operating characteristic (ROC) curve analysis[16] to compare both the predicted risks and the difference between actual age and risk age for each prediction method. ROC curve analysis plots the proportion of individuals correctly classified among those who died (true-positives/deaths = sensitivity) versus the proportion of individuals incorrectly classified among those living (false-positives/survivors = 1 − specificity) for various cut points of the predictor score, i.e., predicted mortality risk or age difference. A center line is called the "chance line" because, for every point, the probability of a true-positive response equals the probability of a false-positive response, i.e., does not predict any better than chance. The greater the area of the curve above this chance line, the greater the predictive accuracy of the instrument (HRA program). Points below the diagonal reflect values of false-positive greater than those of true-positive, i.e, worse results than would be expected by chance alone. Statistical tests were conducted using the Corroc2 program[17] in order to compare the ROC curves of the CDC and Carter Center HRA programs.

TABLE 1—Number (n) of Subjects, Predicted Risk (HRA)[a], Observed 10-Year Mortality Risk (R), and Age-Adjusted 10-Year Mortality Risk (aR)[b] by HRA Method and Difference between Actual Age and Risk Age for Men

Actual Age Minus Risk Age	CDC HRA				Carter Center HRA			
	n	HRA, %	R, %	aR, %	n	HRA, %	R, %	aR, %
>+1[c]	37	1.40	0.0	0.0	464	6.62	8.8	8.0
−1 to +1	212	2.46	0.5	0.5	597	4.20	4.2	3.7
−5 to −1	512	6.00	2.5	2.4	363	6.75	9.6	9.0
−10 to −5	489	16.10	12.3	11.6	62	12.30	21.0	20.3
<−10	257	29.37	17.1	16.4	21	13.32	19.0	17.9
Overall	1507	12.65	7.8	7.4	1507	6.02	7.8	7.2

[a] Average HRA-predicted 10-year mortality risk.

[b] Age-adjusted risks for each sex and HRA method are predicted from the results of four logistic regression models, where each category of risk is transformed into dummy variables and the average risk (−1 to +1) category is left out of the model; age is set equal to 39.6 years (the total sample mean).

[c] Expected to be lowest risk category.

TABLE 2—Number (N) of Subjects, Predicted Risk (HRA)[a], Observed 10-Year Mortality Risk (R), and Age-Adjusted 10-Year Mortality Risk (aR)[b] by HRA Method and Difference between Actual Age and Risk Age for Women

Actual Age Minus Risk Age	CDC HRA				Carter Center HRA			
	n	HRA, %	R, %	aR, %	n	HRA, %	R, %	aR, %
>+1[c]	33	.64	0.0	0.0	84	4.05	1.2	5.0
−1 to +1	576	1.52	0.9	0.9	672	1.98	1.6	2.4
−5 to −1	673	4.45	3.0	2.9	695	4.75	3.0	3.6
−10 to −5	251	10.60	5.2	5.1	126	10.60	8.7	8.4
<−10	95	22.71	10.5	10.4	51	19.84	7.8	7.2
Overall	1628	5.35	2.9	2.9	1628	4.45	2.9	3.6

[a] Average HRA-predicted 10-year mortality risk.

[b] Age-adjusted risks for each sex and HRA method are predicted from the results of four logistic regression models, where each category of risk is transformed into dummy variables and the average risk (−1 to +1) category is left out of the model; age is set equal to 39.6 years (the total sample mean).

[c] Expected to be lowest risk category.

Results

All 3135 never- or current-smoking, 25 to 60 year olds in the 1959 to 1960 Tecumseh Community Health Survey included in this study were White, and 48% were male. Observed mortality risk was 5.3% after 10 years and 13.9% after 20 years.

Correlation of HRA Predictions and Comparison of Difference between Actual Age and Risk Age

Predicted risks of mortality from the two programs were highly correlated (Pearson's $r = .81$; 95% confidence interval (CI) = 0.78, 0.84), as were the risk ages (Pearson's $r = .92$; 95% CI = 0.88, 0.95). These correlations remained high when examined by age group and sex. However, the difference between risk age and actual age (age difference) for the two methods was not correlated (Pearson's $r = .18$; 95% CI = 0.14, 0.21). The average age differences were −0.8 years for the Carter Center HRA and −4.2 years for the CDC HRA. The average age differences between the two programs were 3.4 years (95% CI = 3.2, 3.6) overall, 5.9 years (95% CI = 5.6, 6.2) for men, and 1.1 years (95% CI = 0.79, 1.4) for women. This difference increased when stratified by actual age.

Comparison of 10-Year Mortality Risks by Difference between Actual Age and Risk Age

A risk age that is the same (−1 to +1 category) as actual age signifies that the individual is at an average risk level for his/her age, race, and sex for the CDC HRA (and for Carter Center HRA presence of nonmodifiable risk factors). Thus, we would expect to see an increase in observed mortality risk as the difference between actual age and risk age increases (from > +1 category to < −10 category). The predicted, observed, and age-adjusted mortality risks by age difference are shown in Tables 1 and 2. Overall, the CDC HRA program classifies more people in the higher risk categories than the Carter Center HRA, and thus the predicted mortality risks are almost double those observed among men (12.65% predicted vs 7.8% observed) and among women (5.35% predicted vs 2.9% observed). In contrast, the Carter Center slightly underestimates these risks among men (6.02% predicted vs 7.8% observed) but overestimates risks among women (4.45% predicted vs 2.9% observed).

With the CDC HRA program, as the difference between actual age and CDC HRA risk age increases for both men and women, so do the predicted risks and the total observed and age-adjusted 10-year mortality risks. Using the Carter Center HRA, the lowest age difference (> +1 category) had a higher predicted and observed mortality risk than the next highest category (−1 to +1), after which risks rise steadily for both men and women (Tables 1 and 2).

Relative Operating Characteristic Curve Analysis

We examined ROC curves for both the CDC and Carter Center HRA-predicted risks by 10-year mortality rates, since HRA predictions are based on the

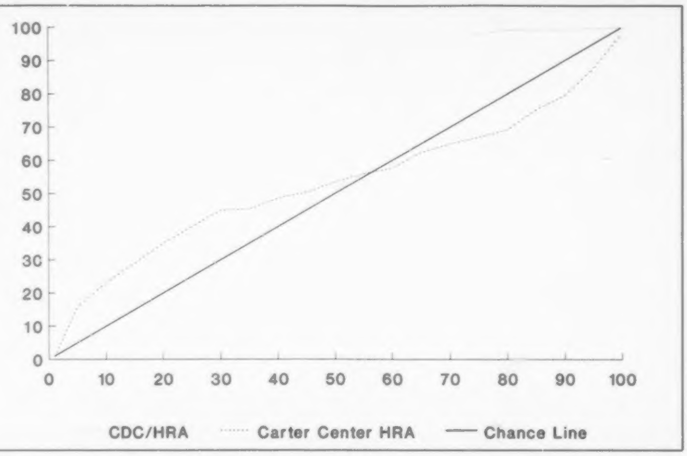

FIGURE 1—ROC Curve Analysis of the Difference between Actual Age and HRA-Predicted Risk Age: Comparison of CDC and Carter Center Methods for *Men*, Using Observed 10-Year Mortality as the Outcome Variable, Tecumseh Community Health Study (n = 1507). Risks were ordered from high to low: CDC difference ranged from −21.5 to 3.7. Carter difference ranged from −16.3 to 7.

10-year appraised chance of dying. The ROC curves for both programs were quite similar and were not significantly different from each other for men ($P = .925$) or women ($P = .9775$). The HRA predictions by age group for each sex were also examined and showed similar results; predictions were best for those under age 45.

The difference between actual age and risk age examined by ROC curves showed that for both men and women (Figures 1 and 2) and within each age group (not shown), actual age minus risk age is a better predictor of mortality with the CDC HRA than the Carter Center HRA. In fact, the ROC curve for the Carter Center HRA was below the chance line for men (Figure 1) but not for women (Figure 2). Thus, for men the Carter Center age difference, on average, predicted 10-year mortality no better than chance alone. As depicted in Figures 1 and 2, these ROC curves were different between the two programs for men ($P = .0083$) and for women ($P = .0257$). Further examination of the data in Figure 1 showed that at the point where the Carter Center HRA crosses the chance line (approximately 60%), the difference between actual age and risk age is −3.6 years, whereas for the CDC HRA it is 1.4 years. Moreover, the mean age for those individuals below the chance line (Carter Center) is 43.3 years, and for the same area of the CDC calculations it is 32.7 years. Those individuals below the chance line for the Carter Center HRA were also more likely to be smokers (99.4%) compared with those before the curve crosses the chance line (79.7%).

We also examined this difference by age groups for the two programs. The results were similar for all ages except for those ages 45 to 60, for whom the Carter Center HRA age difference was consistently below the chance line, indicating that it predicted worse than chance alone for this age group ($P = .0005$).

Discussion

Although mortality risk predictions from the two HRA programs were highly correlated, the calculated risk ages were quite different. It seems unlikely that this result is due to the different reference populations used (the Carter Center program used 1980 to 1982 vital statistics as the reference population and the CDC HRA used 1975 to 1977 vital statistics), because mortality rates have changed little during that time period. More likely, there are differences in how risk age is determined, e.g., mortality risks predicted by the Carter Center more closely approximated those observed than the CDC HRA mortality risks. Further analysis of the specific calculations of risk age is recommended, particularly the implications of using population risk (CDC HRA) versus "adjusted" population risk (Carter Center HRA).

On average, the Carter Center HRA age difference was less than that of the CDC HRA age difference. However, when the age differences for the two programs were compared with the observed 10-year mortality using ROC curve analysis, the CDC HRA age difference was consistently a more accurate (i.e., more sensitive and specific) predictor of individual mortality than was the Carter Center HRA age difference, especially in older men. In addition to performing poorly with the calculation of risk age for older

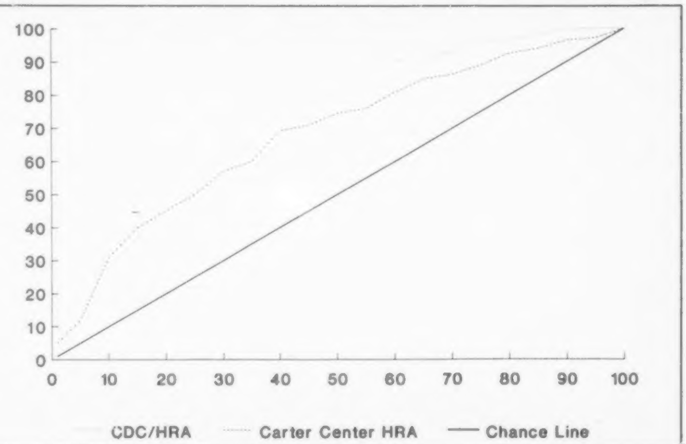

FIGURE 2—ROC Curve Analysis of the Difference between Actual Age and HRA-Predicted Risk Age: Comparison of CDC and Carter Center Methods for *Women*, Using Observed 10-Year Mortality as the Outcome Variable, Tecumseh Community Health Study (n = 1628). Risks were ranked from high to low: CDC difference ranged from −21 to 2.8. Carter difference ranged from −33 to 4.2.

men, the Carter Center HRA may have erred in the weight used for the smoking risk factor.

One of the criticisms of HRA concerns the reliability of self-reported clinical information.[9,10] Reliability was not a problem for blood pressure, weight, and cholesterol in this study, because we used the results of clinical examinations. Although we had data for only approximately 45% of the items used to calculate the predictions (14 of 31 items for CDC/HRA and 13 of 29 items for Carter Center HRA), we did have information on the major risk factors for the common causes of death (e.g., stroke [7.5%], heart disease [39.4%], and lung cancer [5.8%], which accounted for 52.7% of the Tecumseh deaths). Since cardiovascular disease (CVD) is the leading cause of death[18], we also performed ROC curve analysis on the difference between actual CVD risk and predicted CVD risk for each program. The ROC curves for this analysis were virtually identical to those for total mortality; thus the CDC HRA was also a better predictor of CVD mortality.

The variables that we did not have available (e.g., miles traveled per year, social ties) are used as risk factors for less common causes of death, such as motor vehicle accidents and suicide.[8,15] Moreover, we restricted our sample to those individuals older than 25 years of age, and these less common causes of death occur more frequently among younger age groups.[18] Both programs defaulted to population norms for missing values; thus, we would expect the missing items to be extreme to make large changes in the resulting predictions.

Intrinsic errors of the HRA instrument, such as the scientific uncertainty of some risk factors or errors inherent in statistical calculations, will introduce possible problems of accuracy.[15] Our comparison may have been biased toward one program or another because of inherent characteristics of the data used. We attempted to make the comparison as fair as possible by using the same set of variables, calculating ROC curves for 10-year mortality, and using default missing values for each program. Given the constraints of the current software programs and the limitations of the Tecumseh Community Health Study, the CDC HRA appears to be a superior predictor of mortality in the population.

HRA has also been criticized because its effectiveness as a health education/promotion tool has not been demonstrated.[3,8] The revised Carter Center HRA program was primarily developed to update the scientific base of the HRA instrument in order to improve the accuracy of the mortality predictions.[11] If the predictions are improved with this revision, it would seem that this instrument would also be a better health education tool, at least providing more accurate information to the participant. It appears that the revisions did not necessarily improve the mortality predictions (as they were very similar to the CDC HRA mortality predictions) and may have erred on the risk-age calculations for men. If the risk-age calculations are incorrect for a particular program, the effectiveness of that HRA as a health education tool should be questioned. Current HRA users may want to continue using their existing program until the potential problems with the Carter Center Healthier People HRA program are examined further and the effectiveness of the two HRAs in altering behavior has been compared. Those considering the use of the HRA must judge which program better suits the needs of their health education programs. ☐

Acknowledgments

The authors thank Drs. Mark Becker and Richard Cornell for statistical reviews, Dr. Audrey Gomen for editorial assistance, and Dolores Katz for assistance with the statistical analysis, all from the School of Public Health, University of Michigan. This research was funded by the Fitness Research Center, University of Michigan.

References

1. Schoenbach VJ, Wagner EH, Karon JM. The use of epidemiologic data for personal risk assessment in health hazard health risk appraisal programs. *J Chron Dis*. 1983; 36:625–638.
2. Becker MH, Janz NK. Behavioral science perspectives of health hazard/health risk appraisal. *Health Serv Res*. 1987;22:537–551.
3. Schoenbach VJ, Wagner EH, Beery WL. Health risk appraisal: review of evidence for effectiveness. *Health Serv Res*. 1987;22:553–580.
4. Robbins LC, Hall J. *How to practice prospective medicine*. Indianapolis, Ind: Methodist Hospital of Indianapolis; 1970.
5. Goetz AA, McTyre RB. Health risk appraisal: some methodologic considerations. *Nurs Res*. 1981;30:307–313.
6. Safer MA. An evaluation of the health hazard appraisal based on survey data from a randomly selected population. *Public Health Rep*. 1982;97:31–37.
7. Foxman B, Edington DW. The accuracy of health risk appraisal in predicting mortality. *Am J Public Health*. 1987;77:971–974.
8. Kirscht JP. Process and measurement issues in health risk appraisal. *Am J Public Health*. 1989;79:1598–1599.
9. Smith KW, McKinlay SM, McKinlay JB. The reliability of health risk appraisal: a field trial of four instruments. *Am J Public Health*. 1989;79:1603–1607.
10. Smith KW, McKinlay SM, Thorington, BA. The validity of health risk appraisal instruments for assessing coronary heart disease risk. *Am J Public Health*. 1987; 77:419–424.
11. Amler RW, Moriarty DG, Hutchins EB, eds. Healthier people: the Carter Center of Emory University health risk appraisal program guides and documentation. Atlanta, Ga: The Carter Center of Emory University; January 1988.
12. Moriarty DG. *The use and misuse of risk age*. Paper presented at the 24th annual meeting of the Society of Prospective Medicine, St. Petersburg, Fl: Centers for Disease Control; September 24–28, 1988.
13. Centers for Disease Control. CDC health risk appraisal user manual. Atlanta, Ga: CDC Division of Health Education; 1984. GPO publication 746-011/15233.
14. Francis R, Epstein FH. Epidemiological research in a total, natural community, Tecumseh, Michigan. In: Acheson RM, ed. International conference on comparability in epidemiological studies, International Epidemiological Association. *Milbank Q*. 1965;43:333–342.
15. Wiley JA. Predictive risk factors do predict life events. Proceedings of the sixteenth annual meeting. Bethesda, Md: Society for Prospective Medicine; 1980:75–79.
16. Erdreich LS, Lee ET. Use of relative operating characteristic analysis in epidemiology. *Am J Epidemiol*. 1981;114:649–662.
17. Metz CE, Kronman HB. Corroc2 program. Chicago, Ill: Dept of Radiology and Franklin McLean Memorial Research Institute, University of Chicago; April 1980.
18. US Department of Health and Human Services. *Health United States 1987*. Hyattsville, Md; March 1988. DHHS publication 88-1232.

APPENDIX A—CDC vs Carter Center Health Risk Appraisal Program Calculation of an Individual's Overall Predicted Risk of Mortality and Risk Age

CDC HRA	Carter Center HRA
Uses age-sex-race-specific mortality rates for 52 causes of death and "all other" causes	Uses age-sex-specific mortality rates for 41 causes of death and "other causes"
The 10-year expected risk of mortality for each leading cause of death is then "adjusted" by the measured risk factors using the credit-debit method	The individual's current cause-specific mortality risk is estimated using multivariate statistical techniques or population average risk
The cause-specific risks of mortality are summed to determine an overall 10-year expected risk of mortality (predicted risk)	The appraised risks for each cause are summed to get the participant's total appraised risk (predicted risk)
This overall total is compared with age-sex-race-specific risks for the general population to determine "risk age," the age for the given sex-race category with the same overall mortality risk as that predicted from the risk factors	The total appraised risk is compared with either the US population projections for causes of death with modifiable risk factors or an adjusted US population projection for causes of death quantified by nonmodifiable risk factors. This comparison determines the individual's risk age

APPENDIX B—Variables Classified as Precursors and Used Directly by the Carter Center HRA and CDC HRA for Computation of Risk and Those Available from 1959 Tecumseh Community Health Study

Variables Not Available from Tecumseh Study	Variables Available from Tecumseh Study that Required Some Recoding	Variables Available from Tecumseh Study Requiring No Recoding
Smokeless tobacco use[a]	Drinks per week	Age
Miles traveled per year	Hysterectomy	Relative weight
Car size[a]	Diabetes	Blood pressure
Speeding[a]	Cigarettes	Serum cholesterol
Safety belt use	Cigars	Race/origin[b]
Drive after drinking[a]	Pipes	Marital status[b]
Mammogram[a]	Family history of heart disease and diabetes[b]	Sex
HDL cholesterol[a]	Rectal problems[b]	
Pap smear	Lung disease[b]	
Family history of breast cancer		
Menarche[a]		
Age at birth of first child		
Drugs/medication use[b]		
Physical activity level[b]		
Overall health[b]		
Satisfaction with life[b]		
Social ties[b]		
Sleep habits[b]		
Loss/misfortune[b]		
Witness/involved in violent argument[b]		
Risky behaviors[b]		
Breast self-examination[b]		

[a] New variables used by the Carter Center HRA program that were not included in the CDC HRA program calculations.

[b] Variables used by the CDC HRA program that were not included in the Carter Center HRA program calculations.

Impact of a Geriatric Assessment Unit on Subsequent Health Care Charges

ABSTRACT

Background. In a recent randomized controlled trial assessing the efficacy of an inpatient geriatric assessment unit (GAU), we demonstrated that patients assigned to the unit were less likely than those in the usual care group to be admitted to a nursing home and more likely to remain living in the community. Despite evidence of benefits of GAU care, questions remain about its cost. We examine the impact of GAU treatment on subsequent health care charges.

Methods. Sequential referrals of elderly disabled patients considered at risk for institutionalization or likely to improve with rehabilitation were randomized to the GAU or to usual care. Charge data were collected on utilization of a wide variety of health care services starting at the point of randomization.

Results. The group randomized to the GAU experienced significantly higher rehabilitation charges per participant ($1[illegible] 583 vs $2066, $P = .0001$), but lower mean nursing home charges ($1798 vs $3426, $P = .004$). Total health care charges per participant over the 1 year follow-up were greater for the GAU group ($28 406 vs $16 444, $P = .004$). When charges were adjusted per year of life survived, the GAU group still showed a substantial (but nonsignificant, $P = .10$) increase in total charges. However, when charges were adjusted per day subsequently spent residing in the community, adjusted total charges were similar between the two groups.

Conclusion. Our results indicated that improved outcomes from GAU care require an investment in rehabilitation that is not totally offset by decreased institutional charges in the following year. (*Am J Public Health.* 1991;81:1302–1306)

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Introduction

The utility of geriatric assessment has recently been examined by several studies and one National Institute on Aging consensus conference.[1-13] Positive effects of geriatric assessment and subsequent treatment have been demonstrated by Rubenstein and colleagues in a Department of Veterans Affairs Hospital setting and by our group in a community rehabilitation hospital.[14,15] Both of these randomized controlled trials of inpatient geriatric assessment units (GAUs) targeted functionally impaired older patients recovering from acute hospitalization. Both studies demonstrated that GAUs improved patient function, decreased long-term institutionalization, and possibly reduced mortality.[14,15] In our trial, GAU patients were more likely to reside in the community at each follow-up interval.

In our trial, randomization to the GAU or a control group was stratified according to whether the patient was judged prior to randomization to be at higher or lower risk of immediate nursing home placement. When data were analyzed by prerandomization strata, subsequent nursing home days were reduced by GAU treatment for both strata, but physical function was improved and mortality was reduced only in the lower risk stratum.

Despite evidence of benefits of GAU care, questions remain about its cost. In the trial conducted by Rubenstein and colleagues,[14] treatment on the GAU resulted in decreased estimated health care costs per year of life survived. During our study we collected data on both reimbursed and unreimbursed health care utilization charges after randomization and validated the data in an extended data collection project. This article describes the impact of GAU treatment on these charges, both for the entire study cohort and for the two risk strata.

Methods

Patients

Patients who were considered by physicians and social work personnel to be functionally impaired and at risk of nursing home placement were referred to the GAU. Inclusion and exclusion criteria and the screening process have been described previously.[15]

During the 2-year enrollment period 278 referrals were received. Of these, 123 (44%) were determined to be ineligible for the following reasons (percents are of total consultations): medically unstable, 18 (6.5%); terminally ill, 12 (4.3%); too severely cognitively impaired, 34 (12.2%); not sufficiently medically or functionally impaired, 12 (4.3%); refused consent, 21 (7.6%); nursing home placement inevitable, 8 (2.9%); and miscellaneous, 18 (6.5%). The remaining 155 patients were randomized, 77 to the control group and 78 to the GAU group.

Randomization. Randomization was conducted in a stratified block design. Patients were stratified prior to randomization according to an assessment of their risk of immediate nursing home placement at hospital discharge. This assessment

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This paper was submitted to the journal December 3, 1990, and accepted with revisions April 23, 1991.

was based on patient and physician estimate of likelihood of nursing home placement and identified two strata with different levels of risk for subsequent nursing home placement.[15] The methodology for stratification and randomization has been published previously.[15]

The Geriatric Assessment Unit. The GAU studied was a 10-bed geriatric unit based in a rehabilitation hospital that is a separate building in the Baptist Memorial Hospital complex. The organization, specific staff roles, and function of this multidisciplinary unit have been described previously.[2,16] The GAU provided equal emphasis on interdisciplinary assessment of patient problems and on rehabilitation. Particular attention was paid to problems common in frail hospitalized older patients, including incontinence, decubitus ulcers, contractures, confusion, sensory disorders, depression, medication toxicity, inadequate nutrient intake, weakness or lack of motivation, inability to perform activities of daily living (ADL), gait disorders, falls, and degree of family and social support.

After completion of the initial GAU assessments, the first of a series of weekly team meetings was held to determine whether or not the patient was a candidate for a specific treatment and/or rehabilitation. Patients needing medical care were either treated on the GAU or returned to the care of the original referring physician. A rehabilitation plan with specific goals was developed for patients needing rehabilitative care, and patient progress was reevaluated weekly. After discharge from the GAU, patients were returned to their regular physicians and received no further specialized intervention.

Control Group. GAU or research staff made no arrangements for postrandomization care of control group patients. Instead, control group patients received services that they or their physician chose from among options available to any patient, but excluding treatment on the GAU.

Measures

Patient evaluations were made prior to randomization and at 6 weeks, 6 months, and 1 year after randomization. Postdischarge interviews were conducted in the patient's place of residence. All data were collected by trained interviewers who did not provide clinical care but who could not realistically be blinded to group assignment. Data collected included demographic variables, place of residence, self-reported physical activities ranging from basic ADLs to ability to move heavy objects,[17] observed timed physical performance,[18] the CES-D Depression Scale,[19] the Folstein Mini-Mental State Exam,[20] and data on charges.

Charge data were collected on subsequent hospital utilization, physician services, nursing home utilization, inpatient and outpatient rehabilitation, home health care, homemaker/sitter care, medication charges, equipment charges, and other miscellaneous health care-related charges. At the time of randomization, each patient was given a charge data notebook that contained a brief description of the study, a list of possible charges that might be incurred, a chart to record charges, and pockets to store bills, receipts, etc. At the 6-month interview these notebooks were reviewed and, if necessary, patients were reinstructed in charge documentation. At the 1-year interview the notebooks were collected, reviewed with the patient or a family member, and any additional undocumented charges and their sources were noted.

We also constructed a simplified form in layman's language that listed possible categories of health care charges (e.g., hospitalizations, nursing homes, home health care, equipment), asked for the source of these charges (e.g., name of institution, pharmacy, health care agency), and included some open-ended questions such as "Have you been in a nursing home or had someone to care for you in your home?" In cases where a family member or other financial advisor was the responsible party, we mailed the simplified form along with a letter of explanation and a copy of the signed consent form. Follow-up telephone calls were often necessary to verify and explicate charges listed on forms. Telephone calls were also made when forms were not returned, and data were collected from a family member if necessary.

Charge data for all hospital stays, rehabilitation stays, skilled care nursing stays, and home health care were verified with the sources of care. All cooperated, including public and private institutions throughout the United States. Also, length of stay and daily rate were verified with source of care for nursing homes and board and care homes.

For physician, dentist, optometrist, pharmacy, and equipment charges, bills or receipts were used if available. If not, charges were obtained from the source of care. In the few cases where ledger cards of deceased patients had been destroyed, estimates of physician charges were made from the patient's medical record. Medicine charges for which documentation was unavailable were estimated by the patient or family and this estimate was cross-checked with the 1986 *Pharmacy Redbook.*[21] Physician charge data were cross-checked with pharmacy documents to identify otherwise unreported charges.

Because private home care is overlooked in many studies of health care charges, a special attempt was made to collect charge data for private duty nurses, sitters, housekeepers, and paid companions who were hired because of the patient's medical condition. Patients with substantial home care charges generally had a responsible family member who could provide charge data. Also, a large home care registry cooperated fully in providing charge information.

Valid charge data were collected on 152 of 155 randomized patients. Control group data were lacking for one patient who withdrew at 6 weeks and would not allow further data collection and two patients who could not cooperate due to mental status deficits and whose families refused to cooperate.

Statistical Analysis

Equivalence of study groups was tested using Student's t test of differences between means of two independent samples (pooled estimate of population variance) for continuous variables or the chi-square test of statistical independence for categorical variables.[22] All analyses used the intention to treat approach. All statistical tests were nondirectional, with $P < .05$ used as the criterion of statistical significance. Unless specified otherwise, data reported here on utilization and charges represent group means.

Results

The baseline demographic characteristics and functional status of the 152 patients included in this report were similar to those in the study cohort of 155 reported previously.[15] Characteristics of this study cohort included mean age of 78 years, 78% female, 25% married, and 52% living alone. The cohort exhibited substantial functional impairment with 86% rated either moderately disabled (dependent on 3–6 out of 8 ADLs) or severely impaired (dependent on 7 or all ADLs).

There were no significant differences at the time of randomization between the GAU group and the control group in base-

TABLE 1—Absolute Dollar Charges and Charges per Year Survived[a]

Source of Charges	Absolute Dollar Charges			Charges per Year Survived		
	GAU[b]	Control	P^c	GAU[b]	Control	P^c
Rehabilitation units[d]	10 583 ± 6302	2066 ± 4654	.0001	18 406 ± 26 256	2215 ± 4795	.0001
Initial hospitalization[d]	260 ± 1025	1402 ± 3333	.004	279 ± 1081	3148 ± 8868	.005
Subsequent hospitalization[d]	10 401 ± 25 062	5833 ± 8967	.14	33 528 ± 148 157	12 145 ± 23 232	.22
Nursing home	1798 ± 4856	3426 ± 5492	.05	2292 ± 6223	4866 ± 7952	.03
Emergency room, outpatient physician, laboratory	348 ± 563	416 ± 770	.53	392 ± 607	806 ± 2702	.19
Prescription medications	515 ± 443	609 ± 627	.28	539 ± 450	731 ± 765	.06
Home health, housekeeper	3941 ± 10 777	2192 ± 5230	.21	4200 ± 11 383	2744 ± 5729	.32
Other[e]	560 ± 712	501 ± 763	.62	657 ± 862	1478 ± 563	.21
Total	28 406 ± 31 674	16 444 ± 14 504	.004	60 292 ± 159 312	28 134 ± 31 969	.09

[a] Excluding 3 patients with missing data, N=152 (78 GAU, 74 control); data are expressed as mean±standard deviation.
[b] GAU=geriatric assessment unit.
[c] Two-tailed *t* tests for unpaired data.
[d] Postrandomization charges only; includes physician charges.
[e] Equipment, supplies, nonphysician health professionals, and health-related transportation.

line demographic data, scores on the CES-D Depression Inventory, the Mini-Mental State Exam, ADLs, measures of timed physical performance, or types of diagnoses.

Analysis of baseline characteristics of the strata at higher vs lower risk for immediate nursing home placement revealed that the higher risk strata had significantly longer length of initial hospital stay and higher levels of dependence in ADLs at baseline. Age, race, sex, marital status, and prior residence did not differ between the two strata.

Table 1 compares the charges for the GAU vs control groups during the 12 months of follow-up after randomization. The GAU group had significantly higher charges for inpatient rehabilitation (P = .0001) and absolute total charges over the year of follow-up (P = .004). Because admission to the GAU allowed rapid placement, the GAU group had significantly lower hospital charges immediately after randomization (P = .004). The GAU group also incurred significantly lower nursing home charges (P = .05). However, in computing total absolute charges, these lower nursing home and initial hospital charges were not of sufficient magnitude to offset the GAU group's higher rehabilitation charges. As can be seen in Table 1, there were no statistically significant differences between the groups for any other category of absolute charges.

Analysis of the charge data by risk strata (not shown in Table 1) indicated that absolute total charges for the GAU group in the higher risk stratum were significantly higher than those for the corresponding control group for 1 year after randomization ($38 793 vs $16 514 per participant, P = .02). As expected, rehabilitation charges were significantly higher for the high-risk GAU group than for the high-risk control group ($13 118 vs $1888, P = .0001). In the higher risk stratum, mean subsequent nursing home charges tended to be lower for the GAU group ($3078 vs $4826, P = .28) over the year of follow-up, but these differences were not significant and did not offset the overall higher rehabilitation charges. In the lower risk stratum, the GAU group had significantly higher rehabilitation charges compared to the control group ($9164 vs $2168, P = .0001) and the GAU group showed a trend toward higher total charges during the 12-month follow-up period ($22 590 vs $16 404, P = .06). Again, the GAU group in this stratum showed a trend toward lower subsequent nursing home charges ($1081 vs $2621, P = .10), but this trend was not enough to offset overall absolute charges.

Charges were also adjusted per year of life survived to account for persons who died (Table 1). Even when adjusted per year of life survived, the GAU group had significantly higher mean charges than the control group for inpatient rehabilitation ($18 406 vs $2215, respectively; P = .0001). The GAU group had significantly lower adjusted hospital charges immediately after randomization ($279 vs $3148, P = .005) and nursing home charges ($2292 vs $4866, P = .03). The GAU group also demonstrated a trend toward lower medication charges ($539 vs $731, P = .06). However, total adjusted (per year of life survived) charges across all categories were still higher for the GAU group ($60 292 vs $28 134, P = .09), but this difference did not reach statistical significance. However when total charges were adjusted per subsequent day of life spent in the community, the total charges were similar for the two groups ($246 vs $331 per participant).

Discussion

We previously reported that a randomized clinical trial of a geriatric rehabilitation unit in a community rehabilitation hospital had favorable results in allowing elderly persons to continue their residence in a home and demonstrating improved physical function.[15] This article extends those observations by addressing the question: "What are the financial investments required to achieve these outcomes?" Our results indicate that if broadly defined medical care charges are used as the measure of subsequent economic expenditures, improved outcomes require an investment in rehabilitation that is not recovered during the following year. When total unadjusted charges during the 12 months of follow-up were analyzed, a statistically significant increase in absolute total charges for health services was observed for the GAU group. This increase in charges was produced primarily by rehabilitation charges. The mean charge for subsequent nursing home services in the GAU group was less than the comparison group, reflecting the greater likelihood that a patient rehabilitated in the GAU would remain living in the community. However, the difference in mean charges for nursing home services between GAU and comparison groups did not fully offset the greater charges for care in the rehabilitation hospital.

Absolute charges provide one economic perspective, but they can be misleading because patients who die do not generate further health care charges. Also, absolute charges may indicate perspectives of payors but ignore the residential and functional status of the patient. To counteract these problems, findings were also analyzed as charges adjusted per year of life survived and per day of life spent in a community setting subsequent to randomization. Total charges per year of life survived were still substantially higher for the GAU group, but did not reach statistical significance after adjustment. When the total charges for the GAU and control groups were adjusted per day of life spent in a community setting, there was neither a significant difference between the two groups in total charges nor a substantial trend indicating some potential difference. Therefore, although absolute total charges for the GAU group were significantly greater in the year subsequent to randomization, this expense was balanced by the fact that GAU patients had fewer nursing home admissions and were more likely to remain in a community setting.

This study obtained data on actual charges rather than estimating costs. Both approaches have strengths and weaknesses. Health care charges do not necessarily reflect actual costs for delivering a service. On the other hand, estimating costs involves judgments that can have substantial impact on the direction of the ultimate results.[23] Few institutions currently have accurate cost data available. We chose to use actual charges rather than estimated costs because charges more clearly reflect the actual expense of GAU and subsequent care to patients or payors, given current reimbursement methods. A potential limitation of our charge data concerns the reliability of the charge data for noninstitutional services. Items such as housekeeper, home health services, and pharmacy charges may be remembered less reliably. However, we did make every effort to document these categories of charges with source of care and the relatively small magnitude of noninstitutional charges is such that some inaccuracy in these data would not alter the findings of the study.

In the trial conducted in a Department of Veterans Affairs Hospital using estimated costs, GAU patients had similar absolute costs when compared to control patients ($19 406 for the GAU vs $20 329 for the control).[14] When these costs were adjusted per year of life survived, the GAU had lower costs than the control group ($22 597 vs $27 826, respectively). By comparison, in our study, GAU care actually resulted in higher total absolute charges over the 1-year study period. Further, when charges were adjusted per year of life survived, the GAU group still demonstrated a trend toward higher total charges over the year of study, although this difference was not statistically significant. These findings are different from those reported by Rubenstein et al.[14] because our GAU had much higher absolute daily charges. However, higher charges in the community hospital GAU in this study may reflect reimbursement strategies rather than the actual costs essential to mount a successful GAU intervention. Also, our GAU group tended to have higher subsequent hospitalization charges, while the opposite was true in the Rubenstein study. However, all of our study participants were returned to their usual source of care after the GAU while participants in the Rubenstein study continued to receive specialized follow-up care. This difference in design might account for our different findings with regard to subsequent hospital utilization.

The cost-effectiveness data presented here demonstrate an important issue for health policy analysis of services provided to frail older persons. Although absolute charges may be higher for a new form of health care delivery, analysis of charge or cost data should include some form of adjustment per unit of benefit received. Our study found that GAU care led to greater total absolute charges and greater charges per year of life survived. However, when charges were adjusted per day subsequently spent residing in the community, total charges between the two groups were similar. ☐

Acknowledgment

This study was funded by the Robert Wood Johnson Foundation.

References

1. Rubenstein LZ, Abrass IB, Kane RL. Improved care for patients on a new geriatric unit. *J Am Geriatr Soc.* 1981;29:531–536.
2. Applegate WB, Akins D, Vanderzwaag R, et al. A geriatric rehabilitation and assessment unit in a community hospital. *J Am Geriatr Soc.* 1983;31:206–210.
3. Epstein AM, Hall JA, Besdine R, et al. The emergence of geriatric assessment units: the new technology in geriatrics. *Ann Intern Med.* 1987;106:299–303.
4. Lefton E, Bonstelle S, Frengley JD. Success with an inpatient geriatric unit: a controlled study of outcome and follow-up. *J Am Geriatr Soc.* 1983;31:149–155.
5. Allen CM, Becker PM, McVey LJ, et al. A randomized, controlled clinical trial of a geriatric consultation team: compliance with recommendations. *JAMA.* 1986;255:2617–2621.
6. Becker PM, McVey LJ, Saltz CC, et al. Hospital-acquired complications in a randomized controlled clinical trial of a geriatric consultation team. *JAMA.* 1987; 257:2313–2317.
7. McVey LJ, Becker PM, Saltz CC, Feussner JR, Cohen HJ. Effect of a geriatric consultation team on functional status of elderly hospitalized patients. *Ann Intern Med.* 1989;110:79–84.
8. Saltz CC, McVey LJ, Becker PM, Feussner Jr, Cohen HJ. Impact of a geriatric consultation team on discharge placement and repeat hospitalization. *Gerontologist.* 1988;28:344–350.
9. Hogan DB, Fox RA, Badley BWD, Mann OE. Effect of a geriatric consultation service on management of patients in an acute care hospital. *Can Med Assoc J.* 1987; 136:713–717.
10. Williams ME, Williams TF, Zimmer JG, et al. How does the team approach to outpatient geriatric evaluation compare with traditional care: a report of a randomized controlled trial. *J Am Geriatr Soc.* 1987; 35:1071–1078.
11. Yeo G, Ingram L, Skurnick J, Crapo L. Effects of a geriatric clinic on functional health and well-being of elders. *J Gerontol.* 1987;42:252–258.
12. Hendriksen C, Lund E, Stromgard E. Consequences of assessment and intervention among elderly people: a three-year randomised controlled trial. *Br Med J.* 1984;89:1522–1524.
13. NIH Consensus Statement. Geriatric assessment methods for clinical decision making. *J Am Geriatr Soc.* 1987;35:1071–1078.
14. Rubenstein LZ, Josephson KR, Wieland DG, et al. Effectiveness of a geriatric evaluation unit: a randomized trial. *N Engl J Med.* 1984;31:1664–1670.
15. Applegate WB, Miller ST, Graney M, et al. A randomized controlled trial of a geriatric assessment and rehabilitation unit in a rehabilitation hospital. *N Engl J Med.* 1990;322:1572–1578.
16. Applegate WB, Akins DE, Elam JT. A geriatric assessment and rehabilitation unit in a rehabilitation hospital. *Clin Geriatr Med.* 1987;3:145–154.
17. Jette AM, Branch LG. The Framingham disability study: physical disability among the aging. *Am J Public Health.* 1981; 71:1211–1216.
18. Williams ME, Hadler NM, Earp JAL. Manual ability as a marker of dependency in geriatric women. *J Chronic Dis.* 1989;35:115–122.
19. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977;1: 385–401.
20. Folstein MF, Folstein SE, McHugh PR. "Mini-Mental State:" a practical method

for grading the cognitive state of patients by the clinician. *J Psychiatr Res.* 1975;12:189–192.

21. *Drug Topics Red Book.* Cardinale VA (ed.). Oradell, NJ: Medical Economics Publ.; 1986.
22. SPSS, Inc. *SpSS^x User's Guide.* New York: McGraw-Hill; 1983.
23. Avorn J. Benefit and cost analysis in geriatric care: turning age discrimination into health policy. *N Engl J Med.* 1984; 310:1294–1301.

Important Aspects of Right-to-Know Legislation Not Being Used by Communities, Say Tufts Researchers

Although communities can obtain specific information about the manufacture, use, storage, transportation and emission of toxic chemicals within their borders, local planning committees generally have not been using such information to prevent pollution or reduce risks, according to a study done by researchers at Tufts University's Center for Environmental Management.

"An in-depth study of four communities suggests that the right-to-know aspects of the 1986 federal Emergency Planning and Community Right-to-Know Act may be taking a back seat to emergency planning activities," said Kathleen Rest, former project manager and assistant professor of occupational and environmental health at the University of Massachusetts Medical Center. "Such information could be used to persuade local governments and industry to go beyond emergency planning and get involved in accident prevention, risk reduction, and pollution prevention activities," she said.

Congress passed the act, also known as SARA Title III, in 1986, partly in response to the chemical disaster in Bhopal, India, which killed more than 2000 people and injured another 100 000. The law requires communities to establish local emergency planning committees (LEPCs) to develop emergency response plans for such environmental disasters using information previously unavilable to local officials, emergency responders, and citizens.

"So far, LEPCs may be taking a narrow view of their authority and responsibility. These committees could play an important role in prevention, but they will have to be persuaded, and they will need help," Rest said.

The study focused on four communities with different types and levels of industrial activity: Springfield, Mass.; Baytown, Texas; Texas City, Texas; and Newark, New Jersey. The researchers looked at how all local communities involved in the Title III activities obtained, used, interpreted and communicated information about hazardous substances in their communities.

"It's a lost opportunity," said Sheldon Krimsky, principal investigator and professor of urban and environmental policy at Tufts. "The LEPCs could play a more proactive role, but they are going to have to help local citizens understand what the new information means," he said.

The researchers identified specific problems and made recommendations on how to improve the implementation of the law's community right-to-know provisions:

- The US Environmental Protection Agency could organize a task force to discuss realistic roles for the LEPCs and provide guidelines for their involvement in prevention activities.
- LEPCs could be encouraged to be inclusive of all community interests when it appoints community members.
- The LEPCs could share data on accidental releases, chemical inventories, and emissions with public health officials.
- The media could play a significant role in making risk information accessible to the public.
- Grassroots citizen and environmental groups have been successful in motivating citizens to take action about toxic hazards issues. LEPCs should work with these groups to enhance public involvement.
- LEPCs should become forums for constructive interaction between citizens, local government, industry, and the public health community.

"If Congress would provide funding to support state and local community activity, and if states would enact statutes to fund Title III activities through fees or appropriations, then the committees would have some incentive to extend their purview and become more effective advocates for prevention activities," said Krimsky.

Public Health Briefs

Mortality of Intravenous Drug Users in Rome: A Cohort Study

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ABSTRACT

A historical cohort study was carried out in Rome to examine overall and cause-specific mortality among intravenous drug users (IVDUs). A total of 4200 IVDUs (3411 men and 789 women) enrolled in methadone treatment centers between 1980 and 1988 were studied. There were 239 deaths during the follow-up period. The overall SMR was 10.10 in the entire cohort (95% confidence interval, 8.86–11.47), 9.30 in males and 18.07 in females. A large excess of mortality in both sexes was found for infectious, circulatory, respiratory, and digestive diseases as well as for violence, overdose, AIDS, and unknown or ill-defined causes. Tumors and suicide were excessive only in males. Deaths due to drug overdose, violence or trauma, and cirrhosis accounted for 63.6%, AIDS for 7.1%, endocarditis and other bacterial infections for 7.1%, and neoplasms for 3.8% of total mortality. These findings document serious health consequences of drug abuse in Italy. (*Am J Public Health.* 1991;81: 1307–1310)

Introduction

Since the emergence of the acquired immunodeficiency syndrome (AIDS) epidemic most studies have focused on HIV incidence, prevalence, and risk behaviors among intravenous drug users (IVDUs) [1-5] with less attention devoted to overall morbidity and mortality data in this population. There are some indications, however, that IVDUs are at increased risk of overall mortality, deaths from overdose, trauma, infectious diseases.[6-12] This study describes the pattern of mortality in a large cohort of IVDUs in Rome during the period of transition into the "AIDS era."

Methods

Twenty Public Treatment Centers (PTCs) for drug abuse are operating in Rome (population=3 million) since the 1980s. PTCs are open to drug users who reside in each catchment area and agree to a specific outpatient treatment plan. Short-term detoxification with low-dose methadone is currently offered, but subjects usually undertake several methadone cycles. [13]

The three largest PTCs were selected for the study. Information from their clinical records allowed us to enroll a cohort of all the drug users who presented at least once to one of these three centers during the period 1980–1988.

Vital status as of December 31, 1988 was ascertained through the registry office of the last municipality of residence. Cause of death was ascertained through record linkage with the national mortality file for deaths occurring between 1980 and 1987.[14] For deaths occurring in 1988 or not found at the linkage procedure, death certificates were retrieved from the municipality of death and coded by an experienced nosologist according to the ninth revision of the International Classification of Diseases (ICD-9).

For the calculation of person-years at risk of dying,[15] each subject was considered from the date of first enrollment through the end of 1988 or to the date of death. Standardized mortality ratios (SMRs) and their 95% confidence intervals (CI)[16] were used to compare the mortality experience of drug users with that of the total national population. The expected number of deaths was calculated using sex- and age-specific national mortality rates.

Results

A total of 4473 subjects were initially enrolled; 273 were excluded because the identifying data on their records were insufficient to verify vital status, yielding a final sample of 4200 IVDUs (Table 1).

There were only 23.7 expected deaths compared with the 239 observed (SMR-10.10, 95% CI=8.86–11.47) Tables 2 and 3 show sex- and cause-specific mortality. Excess mortality was found for both sexes in all categories. The excess mortality is mainly due to overdose at 3.38 deaths/1000 person-years for both sexes, but violence (1.62/1000 person years) and

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This paper was submitted to the journal September 5, 1990, and accepted with revisions March 6, 1991.

Editor's Note. See related editorial by Peter A. Selwyn on page 1247 of this issue.

TABLE 1—Selected Characteristics of Intravenous Drug User Study Population, Rome, Italy, 1980–1988

	Males (n = 3411)		Females (n = 789)	
Characteristics	No.	%	No.	%
Age of entry into PTC[a]				
<20	428	12.5	150	19.1
20–24	1644	48.2	355	45.2
25–29	981	28.7	191	24.3
>29	361	10.6	90	11.4
Marital Status				
Unmarried	1783	64.1	329	52.7
Married	999	35.9	295	47.3
Education level				
Primary school	821	28.7	79	12.4
Junior high school	1404	49.2	283	44.6
High school	600	21.0	261	41.1
University	31	1.1	12	1.9
Employment status				
Employed	1392	49.4	195	29.2
Unemployed	1428	50.6	474	70.8
Year of entry into PTC				
1980–82	2152	63.0	452	57.5
1983–85	668	19.6	174	22.1
1986–88	594	17.4	160	20.4
Age at first drug use				
<15	85	2.8	35	5.1
15–19	1448	47.7	329	47.6
20–24	1130	37.2	237	34.3
>24	371	12.3	90	13.0
Primary IV drug				
Heroin	3264	96.8	743	95.7
Other	107	3.2	33	4.3
Frequency of use				
Daily	2492	73.1	590	74.8
Less than daily	335	9.8	57	7.2
Unknown	584	17.1	142	18.0
Main treatment				
Methadone	2675	78.4	599	75.9
Psychotropic drugs	398	11.7	86	10.9
Morphine	67	2.0	25	3.2
Others and unknown	116	3.4	28	3.5
None	155	4.5	51	6.5
Vital Status				
Alive	3074	90.1	709	89.9
Dead	200	5.9	39	4.9
Unknown[b]	137	4.0	41	5.2

[a] Public Treatment Center.

[b] Subjects lost to follow-up were considered alive as of December 31, 1988.

cirrhosis (0.88/1000 person years) also contribute. One AIDS death was recorded for the first time in 1986 (0.4/1000 person years); the AIDS death rate increased rapidly in 1987 (7 deaths, 1.6/1000 person years) and 1988 (9 deaths, 2.7/1000 person years).

The SMR for all causes is higher among females than among males even though the excess rates are similar (9.1 and 8.5/1000 person years in males and females, respectively). An increased mortality from suicide is seen only among males; on the other hand SMRs for pneumonia and septicemia are higher in females. Finally, the SMR for malignancies is higher only in males.

Discussion

The study shows that risk of death increased tenfold in male and twentyfold in female IVDUs. The excess risk of death is observed for most causes of death, including neoplasms in males.

Our study population may be different both in character and in exposure to toxic substances from street addicts and occasional users, thus limiting the generalizability of the results. Moreover, IVDUs are a dynamic population, with some current users permanently stopping drug use each year, but we could not take these changes into account because no information was available.

National standards rates were employed to increase the stability of reference numbers, especially in the young population. However, a more restricted reference population, i.e., Rome, would have assured comparability. Therefore, we examined the cause-specific SMRs for 15- to 34-year-olds in the city of Rome compared to Italy during the period 1980 to 1984; SMR values approximate unity for all categories except overdose, endocarditis, and violence with SMRs of 3.7, 1.67, and 0.73, respectively. On that basis, only slightly different results could have occurred using local standards.

We could not control for other potential confounders (i.e., social class, smoking, and alcohol consumption) besides age and sex. Part of the increased risk of death could be explained by the fact that the educational level and the occupational status of our cohort members indicate a lower social class level. However, a study performed in Northern Italy showed that the differences in the overall mortality for social class are not greater than twofold.[17]

Our data agree with published findings about an excess mortality for cirrhosis, endocarditis, septicemia, trauma, suicide, noninfectious heart disease, and AIDS.[6–12, 18,19] Unlike a study performed in New York City, we did not find an excess mortality from tuberculosis[19]; on the other hand, an Italian report showed only a marginally increased risk for tuberculosis among seropositive drug injectors.[20] Females seem more prone to death due to infectious agents (pneumonia and sepsis). We have no explanation for such differences or data to support Stoneburner and colleagues' hypothesis that some of these deaths could be due to undiagnosed HIV-related diseases.[12,19]

Deaths from overdose due to opiates are high. Most of them (72 of 81) occurred when subjects had been out of treatment for more than 2 weeks. It should be noted also that overdose deaths are probably underestimated because some of them are incorrectly ascribed to "ill-defined causes of death" or "other heart diseases."

The excess mortality from neoplasms in males is, to our knowledge, unprecedented in the literature. Even though chance alone could be responsible for this result, smoking and alcohol consumption could be confounding factors, given their strong association with illicit drug use.[21,22] Immunosuppression or even HIV infection might also play a role in cancer development, but this hypothesis requires further investigation.

TABLE 2—Cause-specific Mortality among Male Intravenous Drug Users (19 662 person-years)

Cause of Death[a]	OBS	EXP	SMR[b]	95% CI[c]
All causes (000–999)	200	21.50	9.3	8.1–10.7
Septicemia (038)	2	0.30	67.7	8.2–244.8
Tuberculosis (010–018)	1	0.07	—	—
All malignant neoplasms (140–239)	8	2.97	2.7	1.2–5.3
Nasopharynx (147)	1	0.07	—	—
Digestive organs (150–159)	4	0.58	6.9	1.9–17.6
Stomach (151)	1	0.18	—	—
Colon (153)	2	0.16	12.3	1.5–44.4
Pancreas (157)	1	0.07	—	—
Respiratory organs (160–165)	1	0.42	—	—
Brain (191)	1	0.25	—	—
Lymphatic and hematopoietic tissue (200–207)	1	0.19	—	—
AIDS (279)	16	—	—	—
Overdose (304)	65	—	—	—
Diseases of nervous system (320–389)	2	0.60	3.3	0.4–12.0
Diseases of circulatory system (390–459)	22	2.60	8.5	5.3–12.8
Hypertensive disease (401–405)	2	0.06	31.8	3.9–115.0
Ischemic heart disease (410–414)	1	0.95	—	—
Endocarditis (421–424)	6	0.08	77.1	28.3–165.8
Other forms of heart disease (420;422–423;425–429)	10	0.60	16.5	7.9–30.4
Cerebrovascular disease (430–438)	1	0.60	—	—
Diseases of arteries or veins (460–519)	2	0.30	6.7	0.8–24.1
Diseases of respiratory system (460–519)	3	0.48	6.2	1.3–18.1
Pneumonia (480–486)	2	0.22	9.2	1.1–33.2
Diseases of digestive system (520–579)	20	1.21	16.5	10.1–25.6
Cirrhosis (571)	18	0.81	22.2	13.2–35.1
Injuries and poisoning (800–999)	46	11.58	4.0	2.9–5.3
Suicides (E950–959)	11	1.61	6.8	3.4–12.2
Ill-defined conditions (780–799)	6	0.50	12.1	4.4–26.2
Unknown causes	9	—	—	—

Note OBS = observed deaths; EXP = expected deaths; SMR = standardized mortality ratio; CI = confidence interval.

[a] Code numbers from the International Classification of Diseases, Ninth Revision, 1975.

[b] SMR were not computed for categories with fewer than two deaths.

[c] Two-tailed test.

In conclusion, our findings indicate that drug abuse is an important cause of mortality in the young in Rome as well as in New York City. Preventive and treatment strategies are urgently needed. □

Acknowledgments

This study was partially supported by grant 429, Second Research Project on AIDS (1989), of Ministero della Sanita'-Istituto Superiore di Sanita'.

The authors thank Drs. Mauro Zaccarelli and Andrea Fiume for their help in collecting the data; Drs. Massimo Arca' and Paola Michelozzi for access to the Latium HIV Surveillance System file; Dr. Susanna Crollari for coding; Dr. Carola Tasco for linkage with the mortalilty files; Dr. Elisabetta Magliola for statistical support; Drs. Andrew Moss and Ernest Drucker for the useful discussion; and Profs. Olav Axelson, David Vlahov, Gerry Stimson, and Don C. Des Jarlais for comments on an earlier version of the manuscript. They also wish to thank Dr. Susan Levenstein for editing the manuscript.

Preliminary results of the study were presented at the First International Conference on the Reduction of Drug Related Harm, Liverpool, UK, April 9-12, 1990, and at the Sixth International Conference on AIDS, San Francisco, Calif., June 20-24, 1990.

References

1. Des Jarlais DC, Friedman SR. HIV infection among intravenous drug users: epidemiology and risk reduction. *AIDS.* 1987;1:67–76.
2. Moss AR. AIDS and intravenous drug use: the real heterosexual epidemic. *Br Med J.* 1987;294:389–390.
3. Anderson RM, May RM. Epidemiological parameters of HIV transmission. *Nature.* 1988;333:514–519.
4. Johnson AM. Heterosexual transmission of human immunodeficiency virus. *Br Med J.* 1988;296:1017–1020.
5. Allen J, Curran JW. Prevention of AIDS and HIV infection: needs and priorities for epidemiological research. *Am J Public Health.* 1988;78:381–386.
6. Musto DF, Ramos MR. Notes on American medical history: a follow-up study of the New Haven morphine maintenance clinic of 1920. *N Engl J Med.* 1981;304:1071–1077.
7. Bewley TH, Ben-Aire O, James IP. Morbidity and mortality from heroin dependence. I: Survey of heroin addicts known to Home Office. *Br Med J.* 1968;1:725–726.
8. Wille R. Ten-year follow-up of a representative sample of London heroin addicts: clinic attendance, abstinence, and mortality. *Br J Addict.* 1981;76:259–266.
9. Joe GW, Lehman W, Simpson DD. Addict death rates during a four-year posttreatment follow-up. *Am J Public Health.* 1982;72:703–709.
10. Haastrup S, Jepsen PW. Seven-year follow-up of 300 young drug abusers. *Acta Psychiatr Scand.* 1984;70:503–509.
11. Joe GW, Simpson DD. Mortality rates among opioid addicts in a longititudinal study. *Am J Public Health.* 1987;77:347–348.
12. Selwyn PA, Hartel D, Wasserman W, Drucker E. Impact of the AIDS epidemic on morbidity and mortality among intravenous drug users in a New York City methadone maintenance program. *Am J Public Health.* 1989;79:1358–1362.
13. Sasse H, Salmaso S, Conti S, et al. Risk behaviors for HIV-1 infection in Italian drug users: report from a multicenter study. *J Acquired Immune Defic Syndr.* 1989;2:486–496.
14. Lagorio S, Forastiere F, Michelozzi P, Perucci CA, et al. Cause of death ascertainment in follow-up studies: comparison of procedures available in Italy. *Epidemiol Prev.* 1987;31:57–61.
15. Waxweiler RJ, Beamount JT, Henry JA. A modified life table analysis system for cohort studies. *J Occup Med.* 1983;25:115–124.
16. Rothman KJ, Boice JD. Epidemiologic analysis with a programmable calculator. Washington DC, US Govt Printing Office, 1979. US Dept of Health and Human Services publication (NIH) 79-1649.
17. Costa G, Ciccone G, Vineis P. Mortality by job and economic activity in the Turin longitudinal study. *Epidemiol Prev.* 1989;38:14–29.
18. Dressler FA, Roberts WC. Modes of death and types of cardiac diseases in opiate addicts: analysis of 168 necropsy cases. *Am J Cardiol.* 1989;64:909–920.
19. Stoneburner RL, Des Jarlais DC, Benezra D, et al. A larger spectrum of severe HIV-1 related disease in intravenous drug users in New York City. *Science.* 1988;242:916–919.
20. Antonucci G, Armignacco O, Girardi E, et al. Tubercolosi ed infezione da HIV: incidenza della tubercolosi in soggetti con infezione da HIV e prevalenza di sieropositivita' per HIV-ab in pazienti con tubercolosi. In: *Second Research Project on AIDS (1989): Progress Report.* Rome: ISTISAN Reports 90/10, 1990:133. Ed: GB Rossi.
21. Henningfield JE, Clayton R, Pollin W. Involvement of tobacco in alcoholism and illicit drug use. *Br J Addict.* 1990;85:279–291.
22. De Leon G. Alcohol: the hidden drug among substance abusers. *Br J Addict.* 1989;84:837–840.

TABLE 3—Cause-specific Mortality among Female Intravenous Drug Users (4334 person-years)

Cause of Death[a]	OBS	EXP	SMR[b]	95% CI[c]
All causes (000–999)	39	2.16	18.1	12.9–24.7
Septicemia (038)	2	0.00	509.9	62.0–1842.2
Tuberculosis (010–018)	—	0.01	—	—
All malignant neoplasms (140–239)	1	0.61	—	—
Nasopharynx (147)	—	0.01	—	—
Digestive organs (150–159)	—	0.11	—	—
Stomach (151)	—	0.04	—	—
Colon (153)	—	0.03	—	—
Pancreas (157)	—	0.01	—	—
Cervix uteri (180)	1	0.00	—	—
Respiratory organs (160–165)	—	0.03	—	—
Brain (191)	—	0.03	—	—
Lymphatic and hematopoietic tissue (200–207)	—	0.14	—	—
AIDS (279)	1	—	—	—
Overdose (304)	16	—	—	—
Diseases of circulatory system (390–459)	5	0.37	13.5	4.4–31.6
Hypertensive disease (401–405)	—	0.02	—	—
Ischemic heart disease (410–414)	—	0.06	—	—
Endocarditis (421–424)	1	0.01	—	—
Other forms of heart disease (420;422–423;425–429)	2	0.10	20.8	22.1–655.7
Cerebrovascular disease (430–438)	2	0.12	16.1	1.9–58.2
Diseases of arteries or veins (440–459)	—	0.06	—	—
Diseases of respiratory system (460–519)	3	0.08	37.1	7.6–108.5
Pneumonia (480–486)	3	0.03	88.0	18.1–256.2
Diseases of digestive system (520–579)	3	0.12	24.4	5.0–71.3
Cirrhosis (571)	3	0.07	39.7	8.2–116.0
Injuries and poisoning (800–999)	4	0.60	6.7	1.8–17.1
Suicides (E950–959)	—	0.13	—	—
Ill-defined conditions (780–799)	2	0.04	45.5	5.5–164.5
Unknown causes	2	—	—	—

Note. OBS = observed deaths; EXP = expected deaths; SMR = standardized mortality ratio; CI = confidence interval.

[a] Code numbers from the International Classification of Diseases, Ninth Revision, 1975.

[b] SMR were not computed for categories with fewer than two deaths.

[c] Two-tailed test.

Leptospirosis on Kauai: Investigation of a Common Source Waterborne Outbreak

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ABSTRACT

During the summer of 1987, a common source waterborne outbreak of leptospirosis occurred on the island of Kauai in the state of Hawaii. Eight leptospirosis cases were identified using the microscopic agglutination test. Methodologic weaknesses in this technique were responsible for the initial missed diagnosis on six of the eight cases. All cases had repeated exposure to the Waimea River. A wide array of animal reservoirs and optimal water conditions may have been factors in this outbreak. *(Am J Public Health.* 1991;81:1310–1312)

Introduction

Since the Hawaii State Department of Health (DOH) began collecting detailed statistics on leptospirosis in the early 1970s, Hawaii has consistently had the highest national annual incidence rate.[1] In 1987, the DOH reported a rate of 2.5 per 100 000 population[2] compared with the national rate of 0.02 per 100 000 population.[3] The following report describes a common source outbreak of leptospirosis from recreational exposure to a fresh water source.

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This paper was submitted to the journal October 10, 1990, and accepted with revisions May 6, 1991.

The Outbreak

On July 14, 1987, the DOH was notified of three youths hospitalized with suspected leptospirosis. All had repeated exposures to the Waimea River via swimming. The river runs within the lush Waimea Valley on southwestern Kauai. The valley and hills above the river contain cattle, feral goats, pigs, mongooses, and other rodents.

In April 1987, several spontaneous abortions occurred among a herd of 50 cattle pastured adjacent to the swimming area. Sera from the affected animals (9/9) tested positive for *Leptospira interrogans* serovar hardjo (Sejroe serogroup), with microscopic agglutination test (MAT) titers of 1:800 or greater (personal communication Dr. Craig Nishimoto, 1988, oral).

TABLE 1—Final Microscopic Agglutination Test (MAT) Results

Case No.	Acute Titer	Days after Symptom Onset[a]	Convalescent Titer	Days after Symptom Onset[a]	Presumptive *Leptospira interrogans* Serovar
1	0	21	1:200	62	*australis*
2	1:50	7	1:1600	74	*australis*
3	1:50	4	1:400	61	*australis*[b]
4	1:50	4	1:800	74	*australis*
5	0	3	1:200	25	*bataviae*[c]
6	1:100	3	1:3200	71	*australis*
7	0	0	1:3200	23	*australis*
8	0	1	1:1600	19	*fort bragg*

[a] The number of days after the onset of symptoms each sample was obtained.
[b] Blood culture positive for serovar *bangkok* (Australis serogroup).
[c] Blood culture positive for serovar *bataviae* (Bataviae serogroup).

Methods

Case Finding

The three case-patients and their parents were interviewed in an attempt to identify others with recent river exposure. Sixteen males were identified and sampled, including the three hospitalized youths, who swam at this site almost daily between June 14 and July 4, 1987. Sera were sent to the Leptospirosis Reference Laboratory at the US Centers for Disease Control for MAT evaluation.

A "confirmed" case was defined as an individual with recreational exposure to the Waimea River during the abovementioned dates, and positive leptospiral culture or serologic evidence of recent leptospiral infection manifested by a minimal fourfold increase in MAT titers in paired acute and convalescent sera.

Environmental Investigation

Rodents were trapped at and above the exposure site. Water and mud samples downstream from the swimming spot were cultured for pathogenic leptospires. Seroprevalence data from an island-wide leptospirosis cattle survey conducted in late 1987 by the Hawaii State Department of Agriculture (DOA) were evaluated. Ongoing US Geological Survey measurements on the Waimea River near the exposure site were also evaluated.

Results

Eight individuals reported a flu-like illness with onset between July 8 and July 21, 1987. Symptoms included headache 100% (8/8), fever 100% (8/8), chills 87.5% (7/8), nausea 87.5% (7/8), vomiting 75% (6/8), myalgia 62.5% (5/8), diarrhea 50% (4/8), and jaundice 12.5% (1/8). Five of the eight were hospitalized (62.5%), two were treated as outpatients, and one did not seek medical consultation.

Initial MAT paired sera were positive for only two of the eight symptomatic individuals: one demonstrated *L. interrogans* serovar *bataviae* (Bataviae serogroup), and one demonstrated *L. interrogans* serovar *fort bragg* (Autumnalis serogroup). After blood cultures from an individual with a negative MAT grew *L. interrogans* serovar *bangkok* (Australis serogroup), all sera were retested with a new battery of antigens including serovar *australis* (Australis serogroup), which had been missing from the initial antigen battery. The six with initial negative MAT results demonstrated *L. interrogans* serovar *australis* in the retest (Table 1).

Two cases demonstrated positive blood cultures: one with serovar *bangkok* (as noted above) and one with serovar *bataviae*. The eight asymptomatic males were negative serologically for leptospirosis.

Cases ranged in age from 10 to 24 years (median age=14). The duration of symptoms ranged from 2 to 7 days, with a median of 6 days.

Kidney cultures from 14 rodents trapped near the exposure site as well as water and mud samples from the Waimea River were all culture negative for pathogenic leptospires.

The median river flow rate near the exposure site during the outbreak exposure period was 0.31 m^3/second. The 1987 annual mean flow rate at this site was 1.73 m^3/second. On the two dates most closely bracketing the exposure period, the water temperatures were 22°C and 24°C and the pH measurements were 7.10 and 8.20.[4]

The island-wide DOA cattle seroprevalence survey revealed 89 (64%) of 139 cattle positive for *Leptospira* as manifested by MAT titers of 1:100 or greater against one or more serovars. Of 19 cattle bled on the ranch adjacent to the swimming site, 9 were positive: 8 demonstrated serovar *hardjo*, and 1 demonstrated serovar *bataviae*. Four animals geographically removed from the outbreak demonstrated serovar *australis* (personal communication DOA laboratory report, 1988, written).

Intervention

The local radio station, news media, and press reported the outbreak and warned of the potential danger from swimming in the Waimea River. DOH personnel were on site July 15 and 16 to post warning signs and inform swimmers of the potential danger. In addition individuals obtaining camping or conservation permits were given information on leptospirosis. No subsequent leptospirosis cases were reported from the Waimea River, and only one additional case was reported from the entire island of Kauai during the remainder of 1987.

Discussion

The majority of reported leptospirosis cases both nationally and in Hawaii have been sporadic and unrelated.[5,6] However, cluster outbreaks are reported infrequently.[7-10]

The antecedent epizootic of leptospirosis and the high cattle seroprevalence rate would appear to incriminate cattle as the source of this outbreak. However, because serovar *australis* was not identified in cattle pastured adjacent to the exposure site and because the area contains many potential animal reservoirs, this outbreak may be unrelated to cattle. The small number of feral rodents sampled does not justify exclusion from the analysis.

Optimal water conditions for the survivability of waterborne leptospires—a neutral or slightly alkaline pH[11,12] and temperature of 22°C or higher[13]—were documented in the Waimea River around the outbreak exposure period. Decreased flow rates may have allowed concentration of waterborne *Leptospira*.[10,14] Although river water and mud samples were culture negative, the isolation of pathogenic *Leptospira* by direct culture from soil and natural waters is not routinely done and is generally unsuccessful.[15,16]

Although the MAT is considered a reference test for serological diagnosis of leptospirosis,[17] a negative result does not rule out infection because the patient may be infected with a serotype absent from the battery of testing antigens.[17] This phenomenon was responsible for the initial false negative results on six of eight cases in the present outbreak because serovar *australis* was absent from the testing battery. In addition, the MAT is only partially serovar specific. Cross-reactivity is well documented between serovars in the same serogroup as well as related serovars from different serogroups.[17] This explains the confirmed case with positive blood cultures for serovar *bangkok* but MAT results showing the presumptive presence of serovar *australis*.

Through this experience, we learned that sera from individuals infected with serovar *australis* may not cross-react with antigens from the other serogroups and serovars routinely used in the MAT. To optimize the MAT as a diagnostic tool, the testing battery should include antigens from all leptospiral serovars known to be geographically enzootic. □

Acknowledgments

Grateful acknowledgment is given to Hal Eichelberger, State of Hawaii Department of Health (Kauai), and Dr. Henri Minette, State of Hawaii Department of Health, for their valuable assistance in the investigation and reporting of this outbreak.

References

1. Lewin J. From the Director of Health: leptospirosis in Hawaii. *Hawaii Med J.* 1987;46:330.
2. Hawaii State Dept. of Health, Research and Statistics Office. *Statistical Report, 1988.* Honolulu, HI: 1989.
3. Centers for Disease Control. Summary of notifiable diseases, United States, 1988. Atlanta, GA: 1988. US Dept of Health and Human Services publication (CDC) 90-8017.
4. US Geological Survey, Water Resources Division. *Water Resources Data for Hawaii and Other Pacific Areas, Water Year 1987. Vol. 1, Hawaii.* Springfield, Va: National Technical Information Service, 1988. Report No. USGS/WRD/HD-89/204.
5. Centers for Disease Control. *Leptospirosis Surveillance, Annual Summary 1977.* Atlanta, GA: 1979. US Dept of Health, Education, and Welfare publication (CDC) 79-8260.
6. Anderson BS, Minette HP. Leptospirosis in Hawaii: shifting trends in exposure, 1907–1984. *Int J Zoonoses.* 1986;13:76–88.
7. Havens WP, Bucher CJ, Reimann HA. Leptospirosis: a public health hazard—report of a small outbreak of Weil's disease in bathers. *JAMA.* 1941;116:289–291.
8. Kaufmann AF. Epidemiology trends of leptospirosis in the United States, 1965–1974. In: Johnson RC, ed. *The Biology of Parasitic Spirochetes.* New York: Academic Press; 1976;177–189.
9. Martone WJ, Kaufmann AF. From the Center for Disease Control: leptospirosis in the United States, 1974–1978. *J Infect Dis.* 1979;140:1020–1022.
10. Anderson DC, Follard DS, Fox MD, Patton CM, Kaufmann AF. Leptospirosis: a common-source outbreak due to leptospires of the Grippotyphosa serogroup. *Am J Epidemiol.* 1978;107:538–544.
11. Jacobs R. Leptospirosis—medical staff conference. University of California, San Francisco. *West J Med.* 1980;132:440–450.
12. Hanson LE. Leptospirosis in domestic animals: the public health perspective. *J Am Vet Med Assoc.* 1982;181:1505–1509.
13. Edwards GA, Domm BM. Human leptospirosis. *Medicine.* 1960;39:117–156.
14. Diesch SL, McCulloch WF. Isolation of pathogenic leptospires from water used for recreation. *Public Health Rep.* 1966;81:299–304.
15. Henry RA, Johnson RC. Distribution of leptospira in soil and water. *Appl Environ Microbiol.* 1978;35:492–499.
16. Baker MF, Baker HJ. Pathogenic Leptospira in Malaysian surface waters: I. A method of survey for Leptospira in natural waters and soils. *Am J Trop Med Hyg.* 1970;19:485–492.
17. Sulzer CR, Jones WL, eds. *Leptospirosis: Methods in Laboratory Diagnosis*, Rev. Atlanta, Ga: 1982. Centers for Disease Control; US Dept of Health and Human Services publication (CDC)82-8275.

Timeliness of National Reporting of Communicable Diseases: The Experience of the National Electronic Telecommunications System for Surveillance

ABSTRACT

The timeliness of reporting four nationally notifiable diseases was examined using data reported via the National Electronic Telecommunications System for Surveillance. Timeliness of reporting varied by disease (bacterial meningitis: median 20 days; salmonellosis: median 22 days; shigellosis: median 23 days; and hepatitis A: median 33 days) and by state. These findings indicate a need to standardize surveillance definitions and to account for reporting differences between states in interpreting regional disease trends or detecting multistate disease outbreaks. (*Am J Public Health.* 1991;81:1313–1315)

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Introduction

The timely reporting of communicable diseases to public health authorities is an important part of public health surveillance.[1,2] Prompt reporting permits rapid case investigation and early detection of epidemics. Timeliness is one of the key attributes by which public health surveillance systems should be judged.[3] However, few studies have examined this factor and its potential impact on disease prevention.[4-7]

We determined the delay in reporting of selected communicable diseases using data transmitted from state health departments (SHDs) via the National Electronic Telecommunications System for Surveillance for publication in the *Morbidity and Mortality Weekly Report.*[8] Our findings have implications both for the interpretation of surveillance data and for the design of surveillance systems.

Methods

The Epidemiologic Surveillance Project (ESP) was initiated by the Centers for Disease Control (CDC) in six states in 1984.[8] The system is now referred to as the National Electronic Telecommunications System for Surveillance (NETSS)[1] and receives weekly reports of 49 nationally notifiable diseases from all state and territorial health departments. For this study, NETSS reports were examined for 1987 when 35 SHDs and the District of Columbia participated in the system. The information available for each report included disease, age, race and ethnicity, sex, and the dates of disease onset and report to CDC.

The reporting delay was calculated as the difference in days between date of disease onset and data of report to CDC. Reporting delay was examined for salmonellosis, shigellosis, hepatitis A, and bacterial meningitis (predominantly due to *Neisseria meningitidis* and *Haemophilus influenzae* type B). These diseases were chosen because each may occur in epidemic form so each case report warrants a prompt investigation, and because the diagnosis of each is confirmed by laboratory findings. Reports were analyzed only for states providing the date of disease onset for at least 70% of NETSS cases (Table 1).

Results

The delay in reporting varied markedly by type of disease (Table 1 and Figure 1). The median reporting time ranged from 20 days for bacterial meningitis to 33 days for hepatitis A. More than 75% of reports were made within 8 weeks of disease onset for all diseases. Timeliness of reporting did not differ by the age and race/ethnicity of the patient or by month of disease onset (data not shown). Median reporting delays for salmonellosis and shigellosis varied widely by state, from as little as 4 days for one state to over 8 weeks for another (Figure 2a). The distribution of reporting delays in two states with different patterns in reporting is illustrated in Figure 2b.

Discussion

A principal purpose of public health surveillance of infectious diseases is to prompt public health action in response to changes in disease occurrence.[2] The time-

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This paper was submitted to the journal January 10, 1991, and accepted with revisions February 20, 1991.

TABLE 1—Completeness of Coding of Date of Disease Onset for Selected Diseases, National Electronic Telecommunications System for Surveillance (NETSS) 1987 Disease Reports

	Total NETSS Reports			Selected States with ≥70% Onset Date Coding			
Disease	Number of States Reporting	Total Cases	Percent with Date of Onset Coded	Number of States	Total Cases	Percent with Date of Onset Coded	Median Reporting Delay (days)[a]
Shigellosis	35	9771	78	23	7167	92	23
Salmonellosis	35	25 880	72	22	17 624	92	22
Hepatitis A	35	13 758	74	25	9592	90	33
Bacterial meningitis	33	1497	84	28	1241	94	20

[a] Median time in days from date of disease onset to date of disease report to CDC, for cases with date of onset coded in 1987.

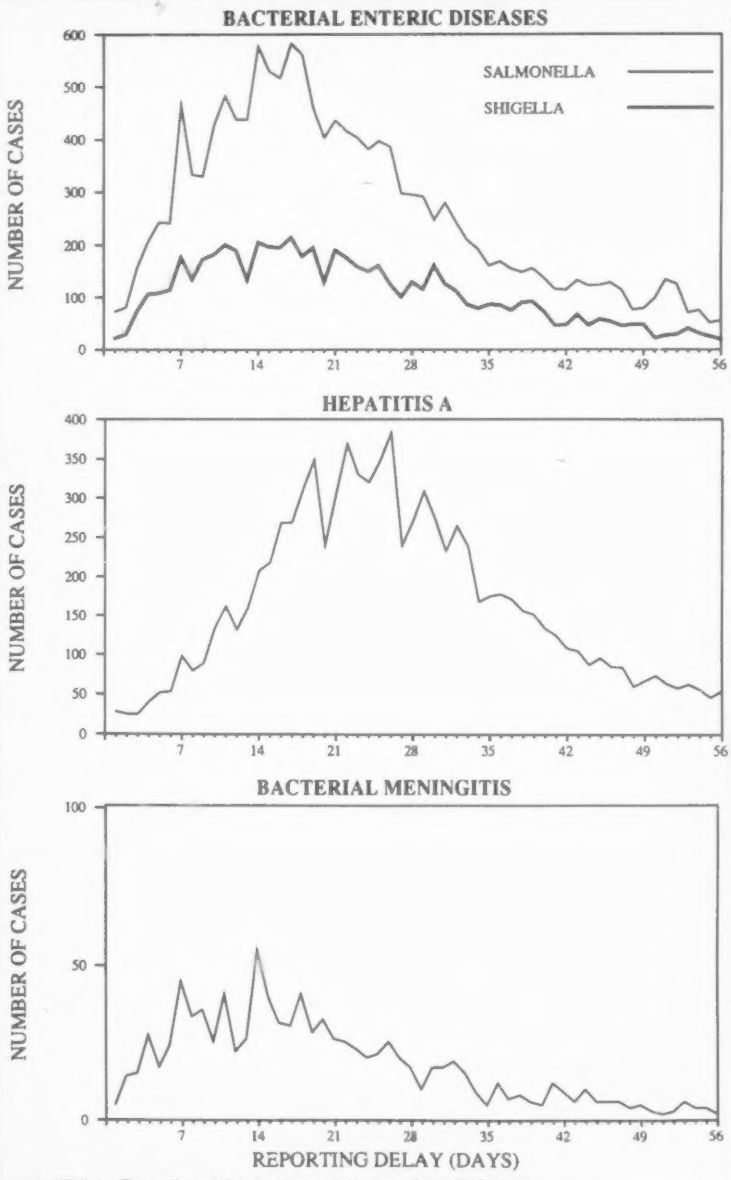

FIGURE 1—Reporting delay by type of disease 1987 ESP data.

liness of reporting is a key element in ensuring that this response is rapid enough to prevent further disease transmission.[1] However, timeliness of reporting and its impact on disease prevention have received little attention in the medical literature.[4–7] We found that the timeliness of reporting to the NETSS system varies widely by type of disease and by state. However, the data available do not allow an evaluation of whether reporting is rapid enough to permit effective disease prevention because timeliness was measured by the report of disease to CDC and immediate preventive action usually takes place at a more local level.

The variability in reporting delay among states may be due to differences among state surveillance systems, including local vs centralized disease reporting, differing statutory requirements for laboratory-based reporting or penalties for failure to report, and differing definitions of disease and of dates of disease onset and disease report. For example, delays in reporting to CDC may be inherent in states in which disease reports go first to county-level health departments before reaching the SHD.

Review of national data does permit detection of regional or multistate disease outbreaks that may be overlooked at the local level. An example of this is the recent recognition of a regional epidemic of *Salmonella* serotype *enteritidis* in the northeastern United States.[9] However, differences in reporting delays between states need to be considered in such an analysis.

Acknowledging the differences in reporting delays by state, the pattern of reporting delays among diseases suggests some factors that may be important in determining reporting time. Reporting of bacterial meningitis was more rapid than for the other diseases studied. This may be due in part to the recognition by physicians and others of the necessity to offer

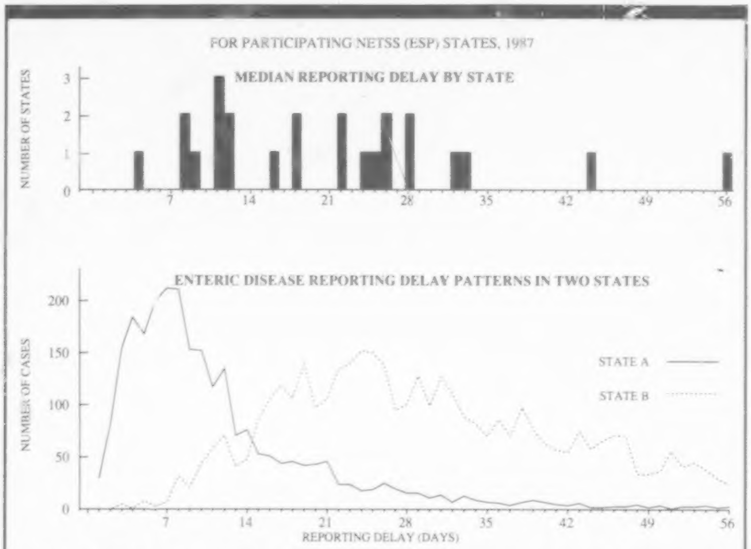

FIGURE 2—Reporting delays from disease onset to report to CDC for enteric disease, by state, for participating National Electronic Telecommunications System for Surveillance States in 1987;
a) = median reporting delay by state;
b) = distribution of reporting delays in two states.

prompt prophylactic treatment with rifampin to close contacts of patients with bacterial meningitis.[10,11] In addition, bacterial meningitis and enteric diseases usually have a more sudden onset and medical care may be sought sooner in the course of disease, allowing more rapid reporting than hepatitis A, which may have a more insidious onset. Finally, bacterial meningitis may be diagnosed from cerebrospinal fluid examination when the patient is first seen by a physician, whereas fecal culture for enteric pathogens and serologic tests for hepatitis A may take several days to complete. This study cannot determine which of these or other factors are most important in determining reporting delay, in part because of the long reporting trail from the physician's office to CDC.

Several areas for improvement in national reporting of surveillance data are highlighted by this analysis. Definitions of date of disease onset and disease report should be standardized. This would parallel recent efforts to standardize disease case definitions and other surveillance system attributes.[3,12,13] For notifiable disease reporting, the date of disease onset is the date of first symptoms attributed to the disease. Date of disease report should be the date of notification of the first-line public health authority responsible for taking immediate preventive action be it town, city, county, or state health department. Health departments should collect the date of disease onset for each reported case. Further studies of the determinants of timely reporting for specific diseases are needed at the local level.

Studies in jurisdictions with unusually short or long reporting times may identify the determinants of the reporting delay. Accounting for inevitable differences in reporting among states in the regional analysis of surveillance data is necessary to take full advantage of national surveillance data for detection of regional or multistate outbreaks. ☐

Acknowledgments

The authors thank Steven Teutsch, MD, MPH, for reviewing the manuscript.

References

1. Thacker SB, Parrish RG, Trowbridge FL. A method for evaluating systems of epidemiologic surveillance. *World Health Stat Q.* 1988;41:11–18.
2. Thacker SB, Choi K, Brachman PS. The surveillance of infectious diseases. *JAMA.* 1983;249:1181–1185.
3. Klaucke DN, Buehler JW, Thacker SB, et al. Guidelines for evaluating surveillance systems. *MMWR.* 1988;37:(suppl. no. S-5); 1-18.
4. Rosenberg ML, Gangarosa EJ, Pollard RA, Wallace M, Brolnitsky O. Shigella surveillance in the United States, 1975. *J Infect Dis.* 1977;136:458–460.
5. Centers for Disease Control. Review of the mechanics of influenza surveillance. In: *Influenza—Respiratory Disease Surveillance.* Report no. 84. September 1968;2–9.
6. Thacker SB, Redmond S, Rothenberg RB, Spitz SB, Choi K, White MC. A controlled trial of disease surveillance strategies. *Am J Prev Med.* 1986;2:345–350.
7. Bernier RH. *Viral Hepatitis Reporting in Greater New Haven.* New Haven, Conn: Yale University School of Medicine, Department of Epidemiology and Public Health; 1974. Thesis.
8. Graitcer P, Burton AH. The epidemiologic surveillance project: report of the pilot phase. *Am J Prev Med.* 1987;3:123–127.
9. St. Louis M, Morse DL, Potter ME, et al. The emergence of grade A eggs as a major source of *Salmonella* serotype enteritidis infections: new implications for control of salmonellosis. *JAMA.* 1988;259:2103–2107.
10. Centers for Disease Control. Update: prevention of *Haemophilus influenza* type b disease. *MMWR.* 1988;37:13–16.
11. Centers for Disease Control. Meningococcal vaccines. *MMWR* 1985;34:255–259
12. Sacks JJ. Utilization of case definitions and laboratory reporting in the surveillance of notifiable communicable diseases in the United States. *Am J Public Health.* 1985;75:1420–1422.
13. Centers for Disease Control. Case definitions for public health surveillance of notifiable diseases. *MMWR.* 1990;39(No. RR-10):1–55.

The Association between Congenital Syphilis and Cocaine/Crack Use in New York City: A Case-Control Study

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ABSTRACT

From 1987 to 1989, an epidemic of congenital syphilis was observed in New York City. A case-control study was done to assess the association between various maternal risk factors and congenital syphilis. Independent of the effect of other factors, the odds of being exposed to cocaine were 3.9 times greater among cases than controls (95% confidence interval, 2.8–5.3). This study suggests that the epidemic of congenital syphilis may be related to the increase in cocaine/crack use among delivering mothers. (*Am J Public Health*. 1991;81:1316–1318)

Introduction

The current epidemic of primary and secondary syphilis among women in New York City (NYC) has been characterized by a doubling in the number of cases between 1985 and 1989[1,2] and has contributed to a secondary epidemic of congenital syphilis. Although fewer than 50 congenital syphilis cases per year were reported to the NYC Department of Health (DOH) between 1980 and 1986, 1017 cases were reported in 1989.[2] During this same period, the proportion of delivering mothers in NYC who reported use of cocaine/crack during pregnancy increased dramatically from 1.0 per 1000 live births to 23 per 1000 live births[3] (Figure 1). Because an association between sexually transmitted diseases and cocaine/crack use had been suggested previously,[4–10] we conducted a case-control study to assess the relationship between various maternal risk factors and congenital syphilis in NYC.

Methods

Congenital syphilis is a reportable disease in NYC, and the NYC DOH maintains a registry of all cases reported from physicians, clinics, and hospitals that meet the revised Centers for Disease Control (CDC) criteria for definite or compatible cases.[11] Additionally, the DOH maintains a birth certificate registry that contains information on all babies born in NYC and on selected characteristics of their mothers.

In the case-control study, all babies reported with congenital syphilis in 1989 were identified from the congenital syphilis registry, and the birth certificate registry was then reviewed to locate their birth certificates. All congenital syphilis babies with available birth certificates were defined as cases. The next two babies born without congenital syphilis on the same day as each case were selected as controls. Cases and controls were also matched on race and hospital of birth. If controls could not be selected on the same date of birth as a case, the subsequent day's births were reviewed until an appropriate control was located. For each of the 14 twin case sets, two controls were selected. All information collected on the babies (race, gestational age, and birth weight) and on their mothers (use of cocaine/crack, heroin, marijuana, methadone, and alcohol; level of prenatal care; age; marital status; level of education; and insurance status) was obtained from the babies' birth certificates.

A bivariate analysis was done to compare cases and controls for each baby and maternal characteristic. The strength of association was evaluated by the odds ratio with 95% confidence intervals; statistical significance was set at the $P < .05$ level. Correlations between variables were examined. Conditional logistic regression analysis was performed to assess the independent association of each of the maternal characteristics with congenital syphilis. Data were analyzed using Statistical Analysis System (SAS) version 5.18.[12]

Results

Of the 987 babies born in NYC listed on the congenital syphilis registry as of March 1990, there were 72 without available birth certificates, 32 stillbirths, and 1 with no available controls. Birth certificates were occasionally not located because of discrepancies in the legal names of the children between the birth certificate and congenital syphilis registries. Of

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This paper was submitted to the journal October 23, 1990, and accepted with revisions May 7, 1991.

the remaining 882 eligible babies, 881 were compatible cases of syphilis and 1 was a definite case.

Cases and controls were matched on race and included non-Hispanic blacks (73%), Hispanics (22%), Whites (4%), and others or unknown (1%). In the bivariate analysis (Table 1), cases had a significantly lower mean birth weight and gestational age than controls (both *P*s < .001). Cases were statistically more likely to use cocaine/crack, alcohol, or other drugs; have no prenatal care; be single; and have less than a high school education (all *P*s < .001). Cocaine/crack use was significantly correlated with alcohol use and being unmarried, and prenatal care was correlated with education (correlation coefficients of .35524, .1988, and .06817, respectively, all *P*s < .001).

Independent of the effect of other factors, the odds of being exposed to cocaine were 3.9 times greater among cases than among controls (Table 2). Other factors significantly associated with congenital syphilis were being unmarried, alcohol use, number of prenatal visits, and having less than a high school education.

Discussion

In this investigation, a strong association was found between congenital syphilis and maternal cocaine/crack use. Moreover, the increase in congenital syphilis cases in NYC since 1985 was temporally related to an increase in cocaine/crack use (Figure 1), whereas the use of alcohol and other drugs remained relatively stable during this period.[3]

Several limitations must be considered in interpreting these data. Underreporting may be a serious problem in examining congenital syphilis in NYC.[13] Drug use information tends to be based on self-reports, and collection of information on the birth certificate is not standardized throughout hospitals. Although the possibility exists that drug histories were more actively pursued among mothers of babies with congenital syphilis, questioning for drug usage is a routine part of medical history-taking done immediately after birth and well before syphilis serologic results are available.

Maternal cocaine/crack use might contribute to the occurrence of congenital syphilis in a variety of ways. Cocaine/crack use might increase the frequency of sexual activity because of the exchange of sex for drugs, or might lead to an increase in unprotected sexual activity.

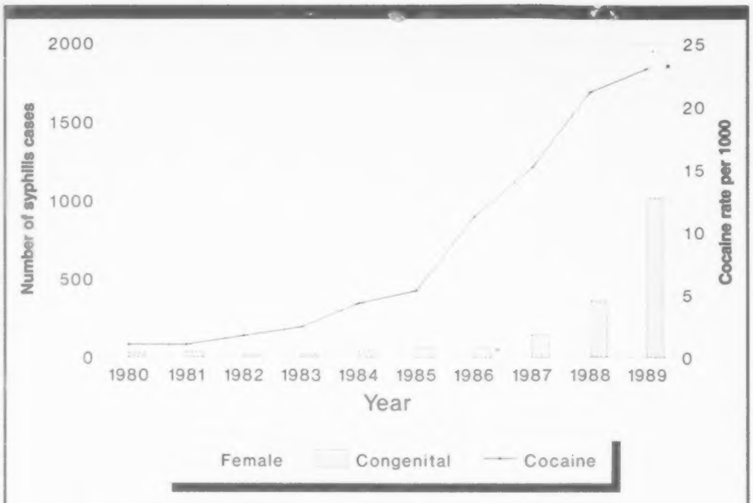

FIGURE 1—Female and congenital syphilis cases and cocaine rate per 1000 live births, New York City, 1980 to 1989

TABLE 1—Comparisons of Cases and Controls: Congenital Syphilis Cocaine/Crack Study, New York City, 1989.

	Cases[a] (n = 882)		Controls[a] (n = 1736)			
Characteristics	No./Total	%	No./Total	%	Crude Odds Ratio	95% Confidence Interval
Babies						
<2500 grams	308/882	35	211/1736	12	3.9	3.2–4.7
<37 weeks gest.	259/732	35	277/1593	17	2.6	2.1–3.2
Mothers						
Cocaine/crack use	249/865	29	93/1710	5	7.0	5.4–9.2
Alcohol use	86/865	10	36/1710	2	5.1	3.4–7.8
Any drug use	403/865	47	262/1710	15	4.8	4.0–5.8
No prenatal care	224/736	30	150/1601	9	5.3	4.1–6.8
1–5 visits	207/736	28	372/1601	23	2.0	1.6–2.5
>5 visits	305/736	41	1079/1601	67	1.0	Reference
Unmarried	748/882	85	1147/1736	66	2.9	2.3–3.6
Not high school graduate	427/834	52	629/1693	37	1.8	1.5–2.1
Age <20 years	95/881	11	251/1730	15	0.7	0.6–0.9
Medicaid	526/858	61	1050/1707	61	1.0	0.8–1.2

[a] Denominators vary because missing data were excluded.

TABLE 2—Variables Significantly Associated with Congenital Syphilis in the Conditional Logistic Regression Model: New York City, 1989

Variable	Regression Coefficient	Adjusted Odds Ratio	95% Confidence Interval
Cocaine/crack	1.354	3.9	2.8–5.3
Unmarried	0.779	2.2	1.7–2.8
Alcohol use	0.723	2.1	1.2–3.4
No. of prenatal visits	−0.098	0.9[a]	0.88–0.93
Less than high school education	0.298	1.3	1.1–1.7

[a] When entered as a continuous variable, with each decreasing prenatal visit, the odds of being a case are increased by 1.1.

Cocaine/crack use could also lead to a decreased use of prenatal care, thereby causing missed opportunities for the detection and treatment of syphilis (38% of case mothers who used cocaine/crack had no prenatal care). The lower socioeconomic status of cocaine/crack-using mothers could be associated with a decreased availability of health services for this population, or with inadequate services. Among women who gave birth to babies with congenital syphilis in NYC who were reported in 1989 and who were documented to have received prenatal care, only a small proportion were documented to have received treatment for syphilis during their pregnancy.[14] Since so few delivering women received treatment for syphilis, treatment failure is probably not an important factor in the increase of congenital syphilis cases.

The provision of accessible and adequate prenatal health care, along with education targeted to women who engage in high-risk sexual activities, is needed to confront the epidemic of congenital syphilis in NYC. ☐

References

1. Centers for Disease Control. Congenital syphilis—New York City, 1986–1988. *MMWR.* 1989;38:825–829.
2. New York City Department of Health. Monthly Morbidity Report, Bureau of Sexually Transmitted Disease Control. New York, NY: New York City Department of Health; 1985–1989.
3. New York City Department of Health. Maternal drug abuse—New York City. *City Health Information.* 1989;8(8):1–4.
4. Goldsmith M. Sex tied to drugs-STD spread. *JAMA.* 1988;260:2009.
5. Fullilove RE, Fullilove MT, Bowser BP, Gross S. Risk of sexually transmitted disease among Black adolescent crack users in Oakland and San Francisco, Calif. *JAMA.* 1990;263:851–855.
6. Andrus JK, Fleming DW, Horan JR, Harger DR, Foster LR. A case-control study of epidemic syphilis, Oregon. In: EIS Conference, Atlanta, Ga; 1989. Abstract.
7. Chaisson RE, Bacchetti P, Osmond D, Brodie B, Sande M, Moss A. Cocaine use and HIV infection in intravenous drug users in San Francisco. *JAMA.* 1989;261:561–565.
8. Centers for Disease Control. Relationship of syphilis to drug use and prostitution—Connecticut and Philadelphia, Pennsylvania. *MMWR.* 1988;37:755–758, 764.
9. Rolfs RT, Golberg M, Sharrar R. Risk factors for syphilis: Cocaine use and prostitution. *Am J Public Health.* 1990;80:853–857.
10. Farley TA, Hadler JL, Gunn RA. The syphilis epidemic in Connecticut: relationship to drug use and prostitution. *Sex Transm Dis.* 1990;17:163–168.
11. Centers for Disease Control. Guidelines for the prevention and control of congenital syphilis. *MMWR.* 1988;37:Suppl. 1:1–13.
12. SAS User's Guide: Statistics. SAS Institute, Inc, Raleigh, NC, 1988.
13. Cohen D, Boyd D, Prabhudas I. The effects of case definition in maternal screening and reporting criteria on rates of congenital syphilis. *Am J Public Health.* 1990;80:316–317.
14. New York City Department of Health. *1989 Congenital Syphilis Year End Report.* New York, NY: New York City Department of Health; 1990:1–21.

Human Papillomavirus, Gonorrhea, Syphilis, and Cervical Dysplasia in Jailed Women

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ABSTRACT

We assessed the prevalence of human papillomavirus (HPV) by cervicovaginal lavage and Southern blot and inquired about behavioral risk factors for cervical disease and sexually transmitted diseases by interview in 114 female detainees at a large New York City jail. Of the women screened, 8% had abnormal Pap smears, 35% had HPV, 7% had gonorrhea, and 22% had serologic syphilis. Given the high rates of HPV infection and cervical cytology, Pap smears should be a routine intake procedure for incarcerated women. (*Am J Public Health.* 1991;81:1318–1320)

Introduction

Prisoners have a high prevalence of drug use, psychiatric disorders, trauma, alcohol abuse, and chronic medical conditions such as respiratory diseases and hypertension.[1,2] The number of incarcerated women in the U.S. increased 53% between 1983 and 1987.[3] At our study site the number of female prisoners has more than doubled in the last 5 years (*SS:unpublished data*).

Prevalence rates as high as 10% for cervical carcinoma in situ, 16% for syphilis, and 5% for gonorrhea have been reported in incarcerated adult women.[4–7] No studies of human papillomavirus (HPV) have been published. Because prisoners have high rates of drug use and prostitution,[3] many are likely to have been exposed to the human immunodeficiency virus (HIV) through these routes.[8] Women

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This paper was submitted to the journal May 14, 1990, and accepted with revisions May 3, 1991.

with HIV may have higher risk of HPV-associated cervical cytologic abnormalities.[9]

We undertook a cross-sectional study of the prevalence of HPV, gonorrhea, syphilis, and cervical cytology in imprisoned women, examined selected behavioral risk factors for HPV and other sexually transmitted diseases (STDs), and looked for any association of HPV with cervical abnormalities in this population.

TABLE 1—Odds Ratios (OR) of HPV Infection and Condom Use among Women with Greater Numbers of Sexual Partners in the Previous Year

Partners, y (n)	OR of Condom Use (95% CI)	OR of HPV Infection (95% CI)
0–1 (54)[a]	1.0	1.0
2–5 (34)	4.1 (0.66–26.1)	1.1 (0.4–2.9)
6–24 (14)	23.7 (6.1–92.1)	2.7 (0.8–9.6)
≥25 (11)	61.5 (13.5–280.3)	1.5 (0.4–5.6)

[a] 0–1 partners = referent group.

Note. ORs are age adjusted; CI = confidence interval; Among women with 25 or more partners in the last year, 73% used condoms and 36% were infected with HPV. Among women with 6–24 partners in the last year, 50% used condoms and 57% were infected with HPV.

Methods

All female detainees at the New York City Correctional Institute for Women undergo an intake examination at the Montefiore Medical Center/Rikers Island Health Service, which provides comprehensive health care to the Rikers Island Jail complex on a contractual basis with the New York City Department of Health. During four nonsuccessive weeks in 1988, eligible female detainees were asked to participate in this study. Eligibility criteria included age less than 50 years, ability to speak English, an intact uterine cervix, history of heterosexual relations, and competency to give informed consent.

The usual medical intake evaluation includes standardized medical history and a physical including pelvic exam and laboratory tests for TB, syphilis, gonorrhea, pregnancy, and a Pap smear. Those who agreed to participate also received a structured interview before the usual intake and cervicovaginal lavage for HPV.[10] The structured interview elicited additional information about sexual and medical histories. Demographic data of participants and nonparticipants were abstracted from the standardized prison medical charts.

All lab analyses were conducted in a blinded fashion. For analytic purposes, normal Pap smears and those showing only atypia with inflammation were considered "no significant pathology"; those classified with atypia and mild dysplasia, or Class III or IV, were considered abnormal in accordance with the Bethesda cytologic classification system.[11] Syphilis serology was evaluated by rapid plasma reagent tests and, if positive, by fluorescent treponemal antibody (FTA) tests. *Neisseria gonorrhoeae* samples were obtained from the endocervix with a cotton swab and cultured on Thayer Martin plates. HPV DNA was extracted from exfoliated cervicovaginal cells collected by lavage. DNA was digested with a *Pst 1* and resultant DNA fragments were separated by gel electrophoresis. HPV DNA was detected by Southern blot analysis with a mixed probe containing radiolabelled HPV 11, 16, 18, and 31 DNA initially under low stringency and following hybridization, under moderate stringency conditions according to techniques previously published.[10] Statistical calculations included odds ratios (ORs) and Mantel-Haenszel $\mathrm{OR}_{\mathrm{MH}}$ to measure strength of association.

Results

Of 145 detainees, 130 met inclusion criteria, and 114 (88% of eligible women) agreed to participate. The most common reason given for refusal was belief that they were not infected; the most common reason for exclusion was a history of hysterectomy. Participants' (N=114) average age was 26.6 years (standard deviation [SD] ± 6.2 years); 53% were Black, 30% Hispanic, and 17% White. Nonparticipants' (N=16) average age was 27.2 years (SD ± 6.8 years); 62% were Black, 25% Hispanic, and 13% White. Sixty-eight percent of the women acknowledged use of some drug, 53% admitted to habitual use of cocaine, and 99% smoked cigarettes.

The median number of reported sexual partners in the last year was 1 (range = 0–2500). Fifty-six percent of the women stated they knew their sexual partners had other sexual partners. The mean age at first coitus was 15 years. Forty women (35%) practiced contraception: 32 used condoms, 5 had tubal ligations, 2 used IUDs and 1 used contraceptive sponges. None used oral contraceptives. Of women using condoms, 80% used them "most" or "all the time." A trend of increased condom use was observed among women with greater numbers of sexual partners (Table 1).

STDs were common: 7% of the women (8/111, 3 missing) had positive gonorrhea cultures, and 22% (24/108, 6 missing) had positive FTA. HPV was the most prevalent STD: 35% of the participants (40/114) were infected.

Pap smears were abnormal in 9% of the women (9/104, 10 missing). There were no cases of invasive cervical cancer. Five of nine women with abnormal Pap smears (56%) were infected with HPV compared to 30 of 95 (32%) women without significant Pap smear pathology (OR = 2.7, 95% confidence interval [CI] = 0.7–10.4). HPV type was not associated with Pap smear abnormality (*R.D. Burk; unpublished data*). The common HPV type prevalence rates were 28% types 16 and 18, 10% type 42, 2% types 6 and 11, and 35% "related" types. Younger women (≤25 years) were more frequently HPV infected (OR = 2.0, 95% CI = 0.9–4.5) and were more likely than older women to have had 2 to 24 sexual partners in the previous year (OR = 2.4, 95% CI = 1.01–5.8). There was an unstable relationship between age and HPV infection when number of sexual partners was controlled for ($\mathrm{OR}_{\mathrm{MH}}$ = 1.8, 95% CI = 0.8–4.1).

Discussion

In 1988, the typical female detainee at Rikers Island was a woman in her mid-20s who engaged in multiple potentially harmful behaviors, including cigarette smoking, cocaine use, sexual activity by age 15, more than one sexual partner in the last year, and low frequency of condom use. The prevalence of STDs and abnormal Pap smears among the women inmates is of a magnitude similar to that of other studies in young inner city and prison women.[12–14] The marked frequency of condom use in highly sexually active women is probably associated with prostitution. The high HPV prevalence may be related to sensitive cell sampling and DNA detection methods.[15] Although this study did not assess incidence through repeated testing, such studies would be desirable.[16]

The majority of these women did not practice safe sex; 72% had not used condoms. These rates of condom use are similar to those reported among juvenile detainees.[13] High STD rates and low use of barrier contraception are particularly disturbing given the prisoners' increased risk of infection with HIV[17,18] and the increased risk of HPV-associated cervical cytologic abnormalities in immunocompromised women.[9,19]

These data suggest the need for universal Pap smear and STD screening of women in prisons and jails, careful follow-up of women with abnormal Pap smears and those who may be HIV positive, and health education for barrier contraception and STD prevention. ☐

Acknowledgments

This research received approval from the Institutional Review Board (IRB) for Human Subjects for the Montefiore Hospital Medical Center and the IRB of the New York City Department of Health. Both IRBs had inmate advocate representation and met federal guidelines for research in jails and prisons. Dr. J.A. Earp provided useful editorial advice. Ms. Carol Spitzer assisted with DNA analyses. Drs. Lutz Gissmann, Harald zur Hausen, and Attila Lorincz provided molecular probes.

This paper was presented in part at the 1990 Southern Regional Meeting of the Society for General Internal Medicine.

References

1. Novick LF, Al-Ibrahim MS. *Health Problems in the Prison Setting. A Clinical and Administrative Approach.* Ill:Bonnerstone House; 1977.
2. Novick LF, Della Penna R, Schwartz MS, Remmlinger E, Lowenstein E. Health status of the New York City prison population. *Med Care.* 1977;15(3):205–216.
3. U.S. Department of Commerce, Bureau of Census. *Law enforcement, courts, and prisons.* In: *Statistical Abstract of the United States 1988*, 108th ed. Washington, DC: 1989:165–174.
4. Moghissi KS, Mack HC, Porzak JP. Epidemiology of cervical cancer: study of a prison population. *Am J Obstet Gynecol.* 1968;100:607–614.
5. Karsai H, Coldman AJ, Gavin D, et al. Cervical intraepithelial neoplasia in female prisoners in British Columbia. *Can Med Assoc J.* 1988;139:733–736.
6. Martin JW, Much DH. Sexually transmitted diseases in prison women. *Penn Med.* 1988;91:40–42.
7. Keighley E. Carcinoma of the cervix among prostitutes in a women's prison. Br J Vener Dis. 1968;44:254–255.
8. Horsburgh CR Jr., Jarvis JQ, McArthur T, Ignacio T, Stock P. Seroconversion to human immunodeficiency virus in prison inmates. *Am J Public Health.* 1990;80:209–210.
9. Feingold AR, Vermund SH, Burk RD, et al. Cervical cytologic abnormalities and papillomavirus in women infected with human immunodeficiency virus. *J Acquir Immune Defic Syndr.* 1990;3:896–903.
10. Burk RD, Kadish AS, Calderin S, Romney SL. Human papillomavirus infection of the cervix detected by cervicovaginal lavage and molecular hybridization: correlation with biopsy results and Pap smear. *Am J Obstet Gynecol.* 1986;154:982–989.
11. National Cancer Institute Workshop. The 1988 Bethesda system for reporting cervical/vaginal cytological diagnoses. *JAMA.* 1989;262:931–934.
12. Rosenfeld WD, Vermund SH, Wentz SJ, Burk RD. High prevalence rate of human papillomavirus infection and association with abnormal Papanicolaou smears in sexually active adolescents. *Am J Dis Child.* 1989;143:1443–1447.
13. Bell TA, Farrow JA, Stamm WA, Critchlow CW, Holmes KK. Sexually transmitted diseases in females in a juvenile detention center. *Sex Transm Dis.* 1985;12:140–144.
14. Alexander-Rodriguez T, Vermund SH. Gonorrhea and syphilis in incarcerated urban adolescents: prevalence and physical signs. *Pediatrics.* 1987;80:561–564.
15. Vermund SH, Schiffman MH, Goldberg GL, Ritter DB, Weltman A, Burk RD. Molecular diagnosis of genital human papillomavirus infection: comparison of two methods used to collect exfoliated cervical cells. *Am J Obstet Gynecol.* 1989; 160:304–308.
16. Reeves WC, Arosemena JR, Garcia M, et al. Genital human papillomavirus infection in Panama City prostitutes. *J Infect Dis.* 1989;160:599–603.
17. Morse DL, Truman BI, Hanrahan JP, et al. AIDS behind bars: epidemiology of New York State prison inmate cases, 1980–1988. *NY State J Med.* 1990;90:133–138.
18. Feldblum PH, Fortney JA. Condoms, spermicides, and the transmissions of human immunodeficiency virus: a review of the literature. *Am J Public Health.* 1988;78:52–54.
19. Vermund SH, Kelley KF, Klein RS, et al. High risk of human papillomavirus infection and cervical squamous intraepithelial lesions among women with symptomatic human immunodeficiency virus infection. Am J Obster Gynecol. 1991 (in press).

Self-disclosure of HIV Infection to Sexual Partners

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ABSTRACT

Self-disclosure of HIV infection to current sexual partners was examined in 138 seropositive men, mostly lower income Hispanics, from Los Angeles. Forty-five percent had been sexually active since learning their serostatus. Of these, 52% had kept their infection secret from one or more partners. The likelihood of disclosure decreased in direct proportion to the number of partners. Nondisclosure occurred in conjunction with unprotected receptive and insertive anal intercourse. Potential factors promoting nondisclosure are discussed. (*Am J Public Health*. 1991; 81:1321–1323)

Introduction

Individuals who are aware they are infected with the human immunodeficiency virus (HIV) and who engage in sexual relations have a social and legal responsibility to disclose their infection to their partners.[1–3] Withholding this information increases the potential for HIV transmission because it creates a situation in which unsafe sexual activity is more likely to occur. Should a partner become infected, he or she may unknowingly infect others. This chain of events suggests that nondisclosure of HIV infection to sexual partners may be an important cause of the continuing HIV epidemic.

Little is known about the prevalence of this behavior. Studies have examined intentions to inform partners among individuals who had not been tested for HIV[4] or had not yet received test results.[5] Two studies have focused on HIV-infected individuals. Of 25 seropositive women, only 52% had disclosed their infection to all sexual partners since diagnosis.[6] One hundred and seven homosexual men informed only 31% of their new sexual partners.[7] We report findings of an ongoing study of self-disclosure in relation to the specific sexual activities performed.

Methods

Adult men who have tested seropositive for HIV in the previous 18 months were recruited for study at a public HIV clinic located in a predominantly Hispanic section of Los Angeles. Study candidates were from any ethnic group, spoke English or Spanish, and were 18 years of age or older.

Recruitment was conducted in the waiting area of the clinic by a bilingual Hispanic woman. Of the men approached, 96% agreed to participate. Each subject completed a self-administered confidential questionnaire in a private section of the waiting area. A four-digit numerical code, uniquely derived by each subject, was used as identification.

Sexual activity and disclosure were measured with a matrix checklist thoroughly explained to each subject. Instructions requested subjects to think of all partners since testing seropositive, including intimate lovers, casual partners, and spouse if married. For each partner, subjects checked the activities that occurred[8,9] and indicated whether they had revealed their HIV infection before the activity (yes = 1; no = 0) and whether the partner was known to be infected with HIV (yes = 1; no or don't know = 0). Subjects who had not had any sexual relations during this time period checked a box at the top of the checklist.

Results

The sample consisted of 138 men (104 Hispanics, 83% of whom used a Spanish language questionnaire; 21 non-Hispanic Whites; 11 Blacks; and 2 Asians). Ninety-one percent identified themselves as homosexual or bisexual. Overall, 13% reported ever injecting a nonprescription drug and 2% reported injecting drugs since they tested seropositive. Seventy percent had an annual income under $10 000.

Sixty-one percent were asymptomatic or had minimal symptoms short of AIDS-Related Complex (ARC), 16% had ARC, and 23% had AIDS.[10] Seventy-five percent had tested HIV seropositive within the previous 8 months.

Sixty-two of the 138 men (45%) had been sexually active (other than kissing) since learning their serostatus. Of these 62, 4 were deleted from the disclosure analysis due to missing data on relevant questionnaire items. Thirty out of 58 men (52%) kept their infection secret from one or more partners. Disclosure did not differ significantly by ethnicity (i.e., Hispanic vs non-Hispanic).

Of the men who had only 1 sexual partner since diagnosis (n = 36), 69% disclosed their serostatus to that partner. Among those with 2 to 4 partners (n = 11),

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This paper was submitted to the journal September 7, 1990, and accepted with revisions February 20, 1991.

TABLE 1—Sexual Activity since Testing HIV Seropositive and Disclosure of HIV Infection to Partners

	Nondisclosure				Disclosure			
	All Partners		HIV-Neg. Partners		All Partners		HIV-Neg. Partners	
Sexual Activity	Ss	Ps	Ss	Ps	Ss	Ps	Ss	Ps
Anal insertive/NC	10	13	10	13	12	13	5	5
Anal insertive/C	12	23	12	22	12	15	9	12
Anal receptive/NC	17	41	17	41	8	8	3	3
Anal receptive/C	16	51	16	48	13	15	10	12
Oral insertive/NC	17	40	14	35	12	15	6	8
Oral insertive/C	5	9	5	9	5	5	4	4
Oral receptive/NC	21	71	21	69	14	15	8	8
Oral receptive/C	7	26	7	26	4	4	4	4
Vaginal insertive/NC	3	3	2	2	0	0	0	0
Vaginal insertive/C	5	15	5	15	4	4	4	4

Note: Ss = no. of subjects; Ps = no. of partners; NC = without condom; C = with condom. Sixty-two men reported sexual activity other than kissing since testing seropositive. Four men from this subgroup had missing data on disclosure and partners' HIV status and were deleted from the analysis. Thus, the entries can be evaluated against 58 men who had sexual relations since testing seropositive and provided full data or against the total sample of 138 men.

36% disclosed to at least 1 partner. Among those with 5 or more partners (n = 11), only 18% disclosed to at least 1 partner. This highly significant trend (χ^2) = 9.50, *P* < .01) indicates that the likelihood of disclosure decreased in direct proportion to the number of partners.

Table 1 displays, for each sexual activity, the total number of subjects, total number of partners, and number of partners presumed to be seronegative at the time of the activity. Information is provided separately for partners to whom subjects disclosed and those to whom subjects did not disclose. Ten subjects (17% of the sexually active) engaged in insertive anal intercourse without using a condom and without disclosing to 13 seronegative partners. Seventeen men (29% of the sexually active) engaged in unprotected receptive anal intercourse without disclosing to 41 seronegative partners. The higher prevalence of receptive than insertive anal intercourse is consistent with a study of HIV-infected homosexual White men from Boston.[11] Both activities are risk factors for transmitting the virus.[12]

In Table 1, the difference between the total number of partners and the number of seronegative partners represents the number of seropositive partners. This difference was larger for informed than uninformed partners, indicating that subjects tended to reveal their infection to those known to be seropositive. When the activity involved insertive or receptive anal intercourse, disclosure to seropositive partners generally occurred in combination with unprotected contact, whereas disclosure to seronegative partners generally occurred in combination with protected contact.

Finally, we examined whether the gender of the partner was associated with disclosure among the bisexual men. Because the activity checklist did not explicitly ask subjects to indicate partner gender, we compared nondisclosure rates for vaginal intercourse (female partners) with nondisclosure rates for receptive anal intercourse (male partners). No significant differences were found.

Discussion

In a sample consisting primarily of lower socioeconomic homosexual/bisexual Hispanic men, we found a relatively high prevalence of nondisclosure of HIV infection to current sexual partners in combination with unsafe sexual activity. For this sample, nondisclosure was not due entirely to lack of knowledge about sexual transmission of HIV. Ninety-four percent of the subjects were aware that they could infect others through unprotected sexual intercourse. Use of alcohol or other nonintravenous drugs before sexual relations, as well as attitudes about social responsibility, may have been factors, although we do not have confirmatory information.

The restricted nature of our sample precludes any wide generalization of the results. Additional studies of Whites, Blacks, Hispanics, and other minority groups of varying socioeconomic levels are needed before a more refined picture can be obtained. Investigation of factors promoting nondisclosure would be immensely valuable for counseling and educational purposes. ☐

Acknowledgments

This research was supported by National Institute of Mental Health grant R01-MH42023. We thank Malcolm C. Pike, PhD, Fred R. Sattler, MD, and Ms. Monica Ruiz for their helpful comments on an earlier version of this paper.

References

1. Gostin L, Curran WJ. Legal control for AIDS: reporting requirements, surveillance, quarantine, and regulation of public meeting places. *Am J Public Health.* 1987;77:214–218.
2. Dicken BM. Legal rights and duties in the AIDS epidemic. *Science.* 1988;239:580–586.
3. Gostin LO. Public health strategies for confronting AIDS. *JAMA.* 1989;261:1621–1630.
4. Temoshok L, Ellmer RM, Moulton JM, Baxter M, Shalwitz J. Youth at high risk for HIV: knowledge, sexual practices, and intentions to inform sexual partners of HIV status. Presented at the 5th International Conference on AIDS, Montreal, Quebec, Canada, June 1989. Abstract T.D.O.29.
5. Kegeles SM, Catania JA, Coates TJ. Intentions to communicate positive HIV-antibody status to sex partners. *JAMA.* 1988;259:216–217.
6. Chervenak JL, Weiss SH. Sexual partner notification: attitudes and actions of HIV-infected women. Presented at the 5th International Conference on AIDS, Montreal, Quebec, Canada, June 1989. Abstract Th.D.P.4.
7. Stempel R, Moulton J, Bacchetti P, Moss AR. Disclosure of HIV-antibody test results and reactions of sexual partners, friends, family, and health professionals. Presented at the 5th International Conference on AIDS, Montreal, Quebec, Canada, June 1989. Abstract E.729.
8. Saltzman SP, Stoddard AM, McCusker J, Moon NW, Mayer KH. Reliability of self-reported sexual behavior risk-factors for HIV infection in homosexual men. *Public Health Rep.* 1987;102:692–697.
9. Coates RA, Calzavara LM, Soskolne CL, et al. Validity of sexual histories in a prospective study of male sexual contacts of men with AIDS or an AIDS-related condition. *Am J Epidemiol.* 1988;128:719–728.
10. Centers for Disease Control. Revision of the CDC surveillance case definition of acquired immunodeficiency syndrome. *MMWR.* 1987;36:3S–14S.
11. McCusker J, Stoddard AM, Mayer KH, Zapka J, Morrison C, Saltman SP. Effects of HIV antibody test knowledge on subsequent sexual behaviors in a cohort of homosexually active men. *Am J Public Health.* 1988;78:462–467.
12. Coates RA, Calzavara LM, Read SE, et al. Risk factors for HIV infection in male sexual contacts of men with AIDS or an AIDS-related condition. *Am J Epidemiol.* 1988;128:729–739.

Immunoglobulins and Growth Parameters at Birth of Infants Born to HIV Seropositive and Seronegative Women

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ABSTRACT

Health characteristics of neonates born to human immunodeficiency virus (HIV) seropositive (n=63) and HIV seronegative (n=57) women were compared. No significant differences were found between the two infant groups for sociodemographic characteristics, mode of delivery, Apgar scores, gestational age, growth parameters, or immunoglobulin levels. Furthermore, no differences were detected in a subgroup of infants later confirmed to have HIV infection (class P-2). Maternal HIV infection does not appear to affect newborn health characteristics. (*Am J Public Health.* 1991;81:1323–1326)

Introduction

As of July 1990, 2464 cases of acquired immunodeficiency syndrome (AIDS) had been identified in patients under 13 years of age,[1] with 78% to 80% presumed to have contracted human immunodeficiency (HIV) infection by vertical transmission.[2,3] The efficiency of virus transmission from an HIV-infected mother to infant is reported to be 13% to 50%.[4-6] Early diagnosis of infection is difficult because transplacentally transmitted anti-HIV antibodies are present in all infants of HIV-seropositive mothers and persist up to 15 months of age or longer.[4,7]

Elevated serum immunoglobulin (IgG, IgM, and IgA) levels are a hallmark of pediatric HIV infection, often developing prior to other laboratory abnormalities such as a decrease in T helper (CD4) cells.[8,9] Birth weight, head circumference, and gestational age have been inconsistently reported to be decreased in infants with perinatal HIV infection.[10-13]

This ongoing prospective study followed a cohort of infants born to mothers at risk for HIV infection to compare the health and growth characteristics of HIV-infected and HIV-noninfected infants from comparable risk strata. The analysis of birth characteristics, specifically growth parameters and immunoglobulins, is presented here.

Methods

All infants born to HIV-seropositive women delivering at an inner-city hospital were eligible for study. An equal number of HIV-seronegative women with acknowledged risk behaviors (intravenous [IV] drug use, sexual contact with IV drug user) were enrolled as control subjects. HIV counseling and testing are offered to all obstetric patients who deliver at this hospital. For most mothers, maternal risk status was determined prenatally by standardized questionnaire and HIV testing performed for consenting women. Newborn HIV testing was offered to unregistered mothers who acknowledged HIV risk factors. Overall there is a 5% seropositivity and 10% refusal rate in this population.[14] In order to examine differences in appropriate intrauterine growth independent of prematurity, infants with less than 34 weeks gestation were excluded.

Each infant's blood was tested within 2 weeks of birth (usually within 24 hours of birth) for HIV antibody and quantitative immunoglobulins, and medical records were reviewed. Weight, length, head circumference, and gestational age were measured in a standardized fashion immediately postdelivery by designated nursery staff. Weight and gestational age by Ballard score[15] was used to classify newborns for intrauterine growth:[16] small, appropriate, or large for gestational age (SGA, AGA, LGA, respectively).

IgG and IgM quantitation was performed by nephelometry (a light scattering technique). Radioimmunodiffusion was used to quantitate IgA levels. The presence of HIV antibody was determined by ELISA (Genetics System, Seattle, Wash) and confirmed by Western blot (DuPont, Wilmington, Del).

Results

Between August 4, 1988, and June 1, 1990, 120 of 127 mothers (95%) agreed to enroll their infant in the study, including 10 infants born to unregistered mothers (5 HIV positive and 5 HIV negative). Sixty-three infants (52.5%) were born to HIV-seropositive mothers, 91% were Black, and 49% were male (Table 1). Maternal substance abuse was the predominant

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This paper was submitted to the journal October 15, 1990, and accepted with revisions April 29, 1991.

TABLE 1—Sociodemographic and Health Characteristics of Infants by HIV Serostatus at Birth (n=120)[a]

Characteristics	HIV Positive (n=63)	HIV negative (n=57)	Total No. (%)
Sociodemographic			
Maternal age (years)	26.2	25.9	26.1
Race			
Black	60	50	110 (91.7)
White or other	3	7	10 (8.3)
Sex of infant			
Male	35	24	59 (49.2)
Female	28	33	61 (50.8)
Risk factor			
IVDA[b]	44	46	90 (75.0)
Sex partner	14	6	20 (16.6)
Multi-risk or unknown	5	5	10 (8.4)
Prenatal Substance Abuse			
Yes	42	48	90 (75.0)*
Health			
Mode of Delivery			
Vaginal	54	47	101 (84.2)
C-section	9	9	18 (15.0)
Missing data			1 (.8)
Apgar score (mean)			
1 minute	7.96	7.98	
5 minute	8.76	8.67	
Gestational age (mean weeks)	38.36	38.66	

[a] Differences between groups for all parameters were nonsignificant at p≥.14 by *t* test and χ^2 test.
[b] IVDA=intravenous drug abuser.
*P=.06.

TABLE 2—Mean Growth Parameters at Birth by Infant HIV Serostatus (n=120)

Parameter	HIV Positive (n=63)	HIV Negative (n=57)	*t* test	*P* Value
Weight (g)	2811	2878	−.67	.51
Length (cm)	47.91	47.94	−.05	.96
Head circumference (cm)	33.23	32.77	1.17	.24
(range)	(28.5–43.5)	(28–37)		

TABLE 3—Mean (range) Immunoglobulin Levels (mg/dl) at Birth by HIV Serostatus

Immunoglobulin	HIV Positive	HIV Negative	*t* test	*P* Value
IgG	1385 (494–2190)	1305 (370–1760)	1.29	.20
IgM	16.4 (6–47)	13.5 (6–59)	1.36	.18
IgA	3.92	3.76	.28	.78
	Normal Values			
	Meites		Fosarelli[b]	
IgG	1086 ± 290		349–1701	
IgM	14 ± 6		16–72	
IgA	2 ± 2		0–13	

[a] Data from Meites S.[16]
[b] Data from Fosarelli et al.[17]

HIV risk factor (75%). Prenatal drug exposure occurred in 84% of infants born to HIV-negative women vs 67% of infants born to HIV-positive women (P=.06).

Type of delivery (85% vaginal) was not statistically associated with seropositivity. Prenatal sexually transmitted disease (gonorrhea and syphilis) exposure was not significant between HIV-positive and HIV-negative infants (P=.24). There was no difference between groups for mean Apgar scores at 1 and 5 minutes or mean gestational age (Table 1).

Fourteen infants (six HIV-positive) were SGA. The majority of these infants (79%) were born to prenatally substance-abusing mothers (HIV-negative =7/8, HIV-positive =4/6; Fisher's exact, P=.39). For all infants, there was no significant difference between SGA and non-SGA infants for prenatal drug use (Fisher's exact, P=.54).

There was no statistically significant difference for any growth parameter between HIV-positive and HIV-negative infants (Table 2). Mean birth weight for HIV-positive infants was 2811 g and 2878 g for HIV-negative infants (range for all infants: 1585–4586 g, P=.51). Three infants (one HIV-positive, two HIV-negative) had a birth weight between 1500 g and 1800 g.

Mean differences for quantitative IgG, IgM, and IgA between the HIV-positive and HIV-negative infants were not statistically significant (Table 3). When mean values for IgG, IgM, and IgA in each group of infants were compared to standardized values previously published[17,18] for a comparable population of Black infants from the same institution,[16] all were within normal limits except for the mean IgM level for HIV-negative infants (13.5 mg/dl), which was slightly lower.

Ten HIV-positive infants developed class P-2 HIV disease[19] during the study period (range of follow-up=9–30 months). Comparison of birth weight, gestational age, and immunoglobulin levels of these 10 HIV-infected infants with remaining HIV-positive infants (P-O, i.e., indeterminate status) and HIV seroreverters (infants born HIV antibody seropositive who reverted to seronegative status by 15–18 months of age) revealed no differences among the three groups (Table 4) with the exception of higher IgA in the HIV reverter group.

Discussion

Our data indicate that in a US urban center with a high prevalence of IV drug use, birth characteristics did not differ between infants born to HIV-positive mothers and HIV-negative mothers with comparable risk. Immunoglobulins were not significantly different between the two groups of infants or between HIV indeterminate, HIV-infected, or HIV seroreverter infants. Elevated immunoglobulin

TABLE 4—Selected Health Characteristics at Birth by HIV Infection Classification[a]

Characteristic	P-2[b] (n=10)	P-O[c] (n=39)	Reverters[d] (n=14)	HIV negative[e] (n=57)	F Test (*P* value)	Difference (*P* value)
Growth						
Birth weight (g)	3049 (2724–3374)	2778 (2612–2945)	2721 (2436–3006)	2878 (1770–4586)	1.35 (.27)	NS
Head circumference (cm)	33.2 (31.7–34.8)	33.2 (32.4–33.9)	33.4 (31.9–34.9)	32.77 (28–37)	.03 (.97)	NS
Gestational age (weeks)	38.9 (37.7–40.1)	38.2 (37.5–38.8)	38.4 (37.3–39.5)	38.7 37.6–38.9)	.52 (.59)	NS
Immunoglobulins (mg/dl)						
IgG	1303 (1049–1556)	1430 (1301–1559)	1324 (1122–1527)	1305 (370–1760)	.65 (.53)	NS
IgM	17.4 (9.4–25.5)	14.04 (9.9–18.1)	21.7 (15.3–28.1)	13.5 (6–59)	2.11 (.13)	NS
IgA	3.3 (.9–5.7)	3.0 (1.9–[illegible].1)	6.6 (4.8–8.4)	3.76 (0–9)	5.91 (.006)	p=.05

[a] Numbers are means and, in parentheses, 95% confidence intervals.
[b] P-2=symptomatic HIV infection.
[c] P-O=indeterminate status (HIV antibody seropositive at birth, asymptomatic <15 months of age).
[d] Reverters=HIV antibody seropositive at birth and converted to HIV seronegative by 15–18 months of age.
[e] HIV negative=HIV seronegative at birth and remained seronegative (controls).

levels have been found by 5 to 9 months of age in HIV-infected infants.[9,20] We found no retardation in growth that could be attributed to maternal HIV infection when infants with similar environmental exposures were compared. Risk factors for HIV infection were dominated by maternal IV drug use or sexual contact with an IV drug user, comparable to trends for HIV infection in women in Western countries.[21–23] Because screening programs targeted to high-risk women fail to identify a substantial proportion of women with HIV infection[14,24] and prenatal HIV testing is voluntary, it is possible that some HIV-exposed infants were not included in this sample. The sample size achieved was sufficient to detect clinically significant differences in weight (265 g), length (1.43 cm), and head circumference (1.38 cm) between HIV-positive and HIV-negative groups with a power of 80%.[25]

Neither set of birth characteristics, growth parameters, or immunoglobulins distinguishes HIV-infected infants from comparable risk noninfected infants at birth. All infants born to HIV-risk women require vigilant pediatric follow-up for early diagnosis of true HIV infection. ☐

Acknowledgments

Funding for this project was provided by National Center for Nursing Research, National Institutes of Health, grant No. NR02069.

References

1. Centers for Disease Control. *HIV/AIDS Surveillance Report.* August 1990.
2. Centers for Disease Control. Update: acquired immunodeficiency syndrome–United States, 1981–1988. *MMWR.* 1989;38:229–236.
3. Katz SL, Wilfert CM. Human immunodeficiency virus infection of newborns. *N Engl J Med.* 1989;320:1687–1689.
4. European Collaborative Study. Children born to women with HIV-1 infection: natural history and risk transmission. *Lancet.* 1991;253–260.
5. Blanche S, Rouzioux C, Moscato MG, et al. A prospective study of infants born to women seropositive for human immunodeficiency virus type 1. *N Engl J Med.* 1989;320:1643–1648.
6. Falloon J, Eddy J, Weiner L, Pizzo PA. Human immunodeficiency virus infection in children. *J Pediatr.* 1989;114:1–30.
7. Pahwa S, Kaplan M, Fikrig S, et al. Spectrum of human T-cell lymphocytic virus type III infection in children: recognition of symptomatic, asymptomatic, and seronegative patients. *JAMA.* 1986;255:2299–2305.
8. Pyun KH, Ochs HD, Dufford MT, Wedgwood RJ. Perinatal infection with human immunodeficiency virus. *N Engl J Med.* 1987;317:611–614.
9. Hutto C, Scott G, Mitchell C, Parks W. Association of laboratory parameters and clinical outcome in human immunodeficiency virus-1 (HIV-1) infected infants. *Pediatr Res.* 1989;25:181A.
10. Checola RT, Hand IL, Noble LM, et al. HIV seropositivity in infants of drug abusing mothers. *Pediatr Res.* 1989;25:276A.
11. Willoughby A, Mendez H, Minkoff H, et al. Prospective longitudinal follow-up of pregnant women infected with human immunodeficiency virus (HIV). *Pediatr Res.* 1989;25:106A.
12. Ryder RW, Wato N, Hassig SE, et al. Perinatal transmission of the human immunodeficiency virus type 1 to infants of seropositive women in Zaire. *N Engl J Med.* 1989;320:1637.
13. Holt E, Boulos R, Halsey NA, et al. The effect of HIV-1 and HTLV-1 infections on birth weight in a population without IV drug abuse. Presented at the Fifth International Conference on AIDS. 1989;997; Montreal, Quebec, Canada: International Development Research Centre. Abstract.
14. Barbacci MB, Dalabetta GA, Repke JT, et al. Human immunodeficiency virus infection in women attending an inner-city prenatal clinic: ineffectiveness of targeted screening. *Sex Transm Dis.* 1990;17:122–126.
15. Ballard JL. A simplified assessment of gestational age. *Pediatr Res.* 1977;11:374.
16. Battaglia FC, Lubchenco LO. A practical classification of newborn infants by weight and gestational age. *J Pediatr.* 1967;71:159–163.
17. Fosarelli P, Winkelstein J, DeAngelis C, Mellits ED. Serum immunoglobulins in the first year of life. *Clin Pediatr.* 1985;24:84–88.
18. Meites S, ed. *Pediatric Clinical Chemistry.* 2nd ed. Washington, DC: American Association for Clinical Chemistry, 1981.
19. Centers for Disease Control. Classification system for human immunodeficiency virus (HIV) infection in children under 13 years of age. *MMWR.* 1987;15:225–236.
20. Welsh G, Hutton N, Modlin J. Anemia and hypergammaglobulinemia in infants with perinatal HIV infection. Presented at the Fifth International Conference on AIDS; 1989;326; Montreal, Quebec, Canada: International Development Research Centre. Abstract.
21. Cowan MJ, Hellman D, Chudwin D, Wara DW, Chang RS, Ammann AJ. Maternal transmission of acquired immune deficiency syndrome. *Pediatrics.* 1984;73:382–386.
22. Rogers MF, Thomas PA, Starcher ET, Noa MC, Bush TJ, Jaffe HW. Acquired immunodeficiency syndrome in children:

report of the Centers for Disease Control national surveillance, 1982 to 1985. *Pediatrics.* 1987;79:1008–1014.

23. Guinan ME, Hardy A. Epidemiology of AIDS in women in the United States: 1981 through 1986. *JAMA.* 1987;257:2039–2042.
24. Landesman S, Minkoff H, Holman S, McCalla S, Sijin O. Serosurvey of human immunodeficiency virus infection in parturients. *JAMA.* 1987;258:2701–2703.
25. Kelsey J, Thompson WD, Evans A. *Methods in Observational Epidemiology.* New York: Oxford University Press; 1986;273–278.

The Impact of HIV on the Usefulness of Sputum Smears for the Diagnosis of Tuberculosis

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ABSTRACT

In a developing country, 289 patients were examined for active pulmonary mycobacterial disease (sputum smear and culture) and HIV infection (serology) to compare the sensitivity and positive predictive value of sputum smears for diagnosing pulmonary tuberculosis in patients with and without antibodies to HIV. Seventy-nine percent of HIV-seronegative vs 66% of HIV-seropositive patients with positive cultures for *Mycobacterium tuberculosis* were smear positive ($P < .05$), and a positive sputum smear predicted the presence of *M. tuberculosis* in 90% of HIV seronegative vs 80% of HIV seropositive patients ($P < .05$). In our opinion, HIV did not significantly compromise the diagnostic utility of the sputum smear. (*Am J Public Health.* 1991;81:1326–1328)

Introduction

The continuing usefulness of sputum smears for the diagnosis of tuberculosis in developing countries has been questioned. In a recent editorial,[1] Pitchenik expressed concern that immunosuppression resulting from human immunodeficiency virus (HIV) type 1 may not only have reduced the sensitivity of the sputum smear by reducing caseation necrosis, and thus the number of acid-fast bacilli in the airway,[2-5] but may also have affected the specificity of the sputum smear by increasing the proportion of patients with nontuberculous mycobacteria.[6] To determine whether HIV has compromised the usefulness of the sputum smear, we compared the sensitivity and positive predictive value of smears for the diagnosis of pulmonary tuberculosis in rural Haitians with and without antibodies to HIV.

Methods

Over a 1-year period beginning March 1988, 323 consecutive rural Haitians presented to a district hospital (Hopital Albert Schweitzer) with suspect pulmonary mycobacterial disease had positive sputum smears for acid-fast bacilli and/or positive cultures for mycobacteria. None had received antituberculous drugs in the preceding 12 months. HIV antibody status was determined in 297 (92%) patients; serum was not available for 4 patients who died shortly after admission and 22 who either refused the test or had already left the facility. Eight HIV-tested children were excluded because the positive culture was from a gastric aspirate and not a sputum specimen. This left a total of 289 (89%) patients for whom results of HIV testing, sputum smear examination, and sputum culture were available.

Two to three fresh, first morning deep cough specimens of sputum were collected from each patient and processed in a manner identical to that reported previously.[7] Standard methods of decontamination, staining, culture, and speciation were applied.[8-11] After obtaining informed consent, serum was collected and tested for antibody to HIV using standard methodology.[12]

Chi-square and Fisher's Exact test were used to compare the sensitivity and positive predictive value of sputum smears for the diagnosis of tuberculosis in HIV-seropositive and HIV-seronegative patients with pulmonary mycobacterial disease.[13]

Results

The 289 study patients ranged in age from 12 to 95 years; 54% were male and 46% female. Table 1 shows that 74 (26%) were HIV seropositive, 222 (77%) smear positive, and 266 (92%) culture positive. Culture was positive for *Mycobacterium tuberculosis* alone in 236, for both *M. tu-*

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This paper was submitted to the journal November 29, 1990, and accepted with revisions April 24, 1991.

berculosis* and nontuberculous mycobacteria (NTM) in 21, and for NTM alone in 9 patients.

It can be deduced from Table 1, that the sputum smear was significantly ($P<.05$) less likely to be positive for acid-fast bacilli in HIV seropositive (50/74, 68%) than in HIV seronegative (172/215, 80%) patients. Likewise, a sputum culture positive for *M. tuberculosis* was less likely ($P=.05$) to be present in HIV seropositive (61/74, 82%) compared to HIV seronegative patients (196/215, 91%). In contrast, HIV seropositive patients were more likely ($P=.05$) to have sputum cultures positive for an NTM alone (5/74, 7%) than were HIV seronegative patients (4/215, 2%). The proportion with a negative culture was not significantly different between HIV seropositive (8/74, 11%) and seronegative (15/215, 7%) patients.

TABLE 1—Sputum Smear and Culture Status of HIV Seropositive and Seronegative Haitians with Pulmonary Mycobacterial Disease

	HIV Positive (n=74)		HIV Negative (n=215)	
Culture	Smear +	Smear −	Smear +	Smear −
TB	37	18	143	38
TB and NTM	3	3	12	3
NTM	2	3	2	2
Negative	8	0	15	0
Total	50	24	172	43

Note. HIV = human immunodeficiency virus type 1; TB = *M. tuberculosis*; NTM = nontuberculous mycobacteria; + = positive; − = negative.

TABLE 2—Species of NTM Isolated from HIV Seropositive and Seronegative Haitians with Pulmonary Mycobacterial Disease

	HIV Positive				HIV Negative				
	Smear +		Smear −		Smear +		Smear −		
	TB Culture		TB Culture		TB Culture		TB Culture		
NTM	+	−	+	−	+	−	+	−	Total
M. scrofulaceum	2	2	1	1	6		1		13
M. gordonae					4		2		6
M. fortuitum				1[a]		2[b]		2[a]	5
M. flavescens			1		1				2
M. avium complex					1				1
M. vaccae			1						1
M. avium complex M. gordonae	1								1
M. gordonae and *M. fortuitum*				1[a]					1
Total	3	2	3	3	12	2	3	2	30

Note. HIV = human immunodeficiency virus type 1; NTM = nontuberculous mycobacteria; TB = *M. tuberculosis*; + = positive; − = negative.

[a] Biovariant *peregrinum*.

[b] Biovariant *fortuitum*.

The sensitivity of a positive acid-fast smear to identify subjects with a positive *M. tuberculosis* culture was significantly greater in HIV seronegatives than in HIV seropositives. The sensitivity in HIV seronegatives was 79% (155/196) in comparison to 66% (40/61) in HIV seropositives. In addition, a positive sputum smear predicted the presence of *M. tuberculosis* in 80% (40/50) of HIV seropositive patients and 90% (155/172) of HIV seronegative patients, a difference that was also statistically significant ($P<.05$). Neither the sensitivity nor the positive predictive value varied by age or sex.

The species of NTM isolated from 30 patients are shown in Table 2. *M. scrofulaceum* was the most common NTM isolated (13/30, 43%). In 10 instances it was isolated together with *M. tuberculosis* and in 3 it was the only isolate. All of the latter isolates were from HIV seropositive patients. *M. fortuitum* was the next most common NTM isolated (6/30, 20%). In each instance (except one where it was isolated together with *M. gordonae*) it was the only isolate, accounting for 67% (6/9) of the patients in which an NTM alone was isolated. Of the 6 patients from whom *M. fortuitum* was isolated, 2 were HIV seropositive.

Discussion

Particularly in developing countries, where resources for mycobacterial culture are often not available, acid-fast smears are relied on to diagnose tuberculosis. Before the AIDS epidemic the sensitivity of a positive sputum smear was relatively low, but the positive predictive value was high.[14] A patient with a positive smear was almost certain to have tuberculosis and was treated accordingly. Whether HIV modifies the sensitivity and positive predictive value of the sputum smear, especially in developing countries where HIV seroprevalence may be high,[15] is not known. In our study of rural Haitians we found small but significant differences in the sensitivity and positive predictive value of sputum smears between HIV seropositive and HIV seronegative pulmonary tuberculosis patients; 79% of HIV seronegative vs 66% of HIV seropositive patients with positive cultures for *M. tuberculosis* were smear positive, and a positive sputum smear predicted the presence of *M. tuberculosis* in 90% of HIV seronegative vs 80% of HIV seropositive patients. In our opinion, these differences are small and do not significantly compromise the usefulness of sputum smears in developing countries such as Haiti, where the prevalence of tuberculous infection is high.[16] Regardless of HIV status, patients with sputum smear positive pulmonary mycobacterial disease should be assumed to have disease caused by *M. tuberculosis* and should be treated accordingly. ☐

Acknowledgments

The authors thank George Carre, MD, and Roosevelt Occenac of Hopital Albert Schweitzer; Shirley Parker, MD, Joyce Wolfe, and Brenda Binda of the Mycobacteriology Reference Laboratory Health Sciences Centre, Winnipeg, Manitoba; Albert Laszlo, PhD, and Dorothy Helbecque of the Laboratory Centre for Disease Control, Ottawa, Ontario, Canada; and Eileen Walters and Heather Smith for the preparation of the manuscript. This work is dedicated to the memory of William Larimer Mellon, Jr. (1910–1989), who together with his wife Gwen, founded Hopital Albert Schweitzer in Haiti.

The research for this paper was supported in part by grants from the Manitoba Lung Association and the Discretionary Funds of the Dean, Faculty of Medicine, and the Chair, Department of Medicine, University of Manitoba.

References

1. Pitchenik AE. Tuberculosis control and the AIDS epidemic in developing countries. *Ann Intern Med.* 1990;113:89–91. Editorial.
2. Pitchenik AE, Fertel D, Block AB. Mycobacterial disease: epidemiology, diagnosis, treatment, and prevention. *Clin Chest Med.* 1988;9:425–441.
3. Klein NC, Duncanson FP, Lenox TH, Pitta A, Cohen SC, Wormser GP. Use of mycobacterial smears in the diagnosis of pulmonary tuberculosis in AIDS/ARC patients. *Chest.* 1989;95:1190–1192.
4. Dannenberg AM Jr. Immune mechanisms in the pathogenesis of pulmonary tuberculosis. *Rev Infect Dis.* 1989;11(suppl 2):S369–S378.
5. Grosset J. Bacteriologic basis of short course chemotherapy for tuberculosis. *Clin Chest Med.* 1980;1:231–241.
6. Sathe SS, Reichman LB. Mycobacterial disease in patients infected with the human immunodeficiency virus. *Clin Chest Med.* 1989;10:445–463.
7. Scalcini M, Carre G, Jean-Baptiste M, et al. Antituberculous drug resistance in central Haiti. *Am Rev Respir Dis.* 1990;142:508–511.
8. Sommers HM, Good RC. Mycobacterium. In: Lennette EH, Ballows A, Hausler WJ Jr, Shadowy HJ, eds. *Manual of Clinical Microbiology.* 4th ed. Washington, DC: American Society of Microbiology; 1985: 216–248.
9. Snider DE Jr., Good RC, Kilburn JO, et al. Rapid drug susceptibility testing of *Mycobacterium tuberculosis. Am Rev Respir Dis.* 1981;123:402–406.
10. Heifets LB. Rapid automated methods (Bactec system) in clinical mycobacteriology. *Semin Respir Infect.* 1986;1:242–249.
11. Vestal AL. Procedures for the isolation and identification of mycobacteria. Washington, DC: US Govt Printing Office, 1975. US Dept of Health, Education, and Welfare publication (CDC) 76–8230.
12. Long R, Scalcini M, Manfreda J, et al. Impact of human immunodeficiency virus type-1 on tuberculosis in rural Haiti. *Am Rev Respir Dis.* 1991;143:69–73.
13. Statistical methods in epidemiology. In: Armitage P, Barry G, eds. *Statistical Methods in Medical Research.* 2nd ed. London, England: Blackwell Scientific Publications; 1987:455–484.
14. Daniel TM. Rapid diagnosis of tuberculosis: laboratory techniques applicable in developing countries. *Rev Infect Dis.* 1989:2(suppl 2):S471–S478.
15. Pape JW, Johnson WD Jr. Epidemiology of AIDS in the Caribbean. *Bailliere's Clin Trop Med and Commun Dis.* 1988;3:31–42.
16. Pitchenik AE, Russell BW, Cleary T, Pejovic I, Cole C, Snider DE Jr. The prevalence of tuberculosis and drug resistance among Haitians. *N Engl J Med.* 1982; 307:162–165.

Human Exposure to Rabies from Pet Wild Raccoons in South Carolina and West Virginia, 1987 through 1988

Bradley A. Woodruff, MD, MPH, Jeffrey L. Jones, MD, MPH, and Thomas R. Eng, VMD, MPH

ABSTRACT

During 1987 and 1988, exposures to eight pet wild raccoons in South Carolina and West Virginia resulted in administration of rabies postexposure prophylaxis to 19 children and 26 adults. All eight raccoons appeared normal at the time of capture, and three had no signs of illness when sacrificed. The direct medical cost resulting from these exposures was $23 714 ($527 per person). Regulations and public education may help decrease this type of rabies exposure. (*Am J Public Health.* 1991;81:1328–1330)

Introduction

Although human rabies deaths in the United States are extremely rare, approximately 18 000 persons per year receive rabies postexposure prophylaxis (CDC, unpublished data). In most southeastern and mid-Atlantic states, including South Carolina and West Virginia, raccoons account for the largest proportion of laboratory-confirmed rabid animals.[1] Most human exposures from rabid raccoons and other wild animals involve animals encountered in the wild, but exposures to rabid wild animals kept as pets have been documented.[2–4] As a result, several health organizations and government agencies recommend that wild animals not be kept as pets, and most states have laws requiring special permits to keep such animals[5]; however these measures may be ineffective. In this report, we describe eight incidents where wild raccoons were captured and kept as pets resulting in potential human exposure to rabies in South Carolina and West Virginia in 1987 and 1988.

Methods

State public health personnel in West Virginia and South Carolina were interviewed regarding incidents of possible human rabies exposure resulting from pet wild raccoons during 1987 and 1988. All reports were further investigated by interviewing exposed persons, physicians, and public health personnel and by reviewing records in state laboratories, physicians' offices, and emergency rooms. Data were available only for persons who received prophylaxis.

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This paper was submitted to the journal November 19, 1990, and accepted with revisions April 24, 1991.

Human exposure to rabies was defined as a bite, a scratch, or direct contact with saliva from a laboratory-proven rabid animal. Potential exposure was defined as similar contact with an animal that was not tested.

Direct medical care costs included the cost of rabies immune globulin (RIG) and human diploid cell vaccine (HDCV) and the costs for services provided by hospitals and physicians. Medical costs were calculated from billed amounts. Direct social costs, such as the costs of contact investigations and laboratory testing, and indirect personal costs, such as lost wages and transportation costs, were not included in cost estimates because data were unavailable.

Results

During 1987 and 1988, 8 incidents occurred in South Carolina and West Virginia involving potential or actual human rabies exposure from pet wild raccoons (Table 1). None of the eight raccoons had signs of rabies or behaved abnormally at the time of capture. The median interval between capture and the discovery of rabies was 1 month (range = 7 days to 1 year). The precise incubation period was unknown for these animals; however, minimum incubation periods could be derived from the date of last possible contact with other potentially rabid animals.

Six raccoons were tested for rabies; five were positive and one was reported as "inconclusive." Of the five positive animals, two appeared normal at the time of sacrifice, two died after developing signs of illness, and one died suddenly without signs of illness. Two animals were not tested for rabies: one was shot in the head, making testing impossible, and the other escaped after biting the owner. Of the three animals sacrificed without signs of illness, two were killed only after persons who had been bitten demanded that the animals be tested.

Of the 45 persons receiving prophylaxis, 19 (42%) were children ≤ 15 years of age. Eighteen (40%) persons were bitten, 25 (56%) were scratched or exposed to saliva, and 2 (4%) had no apparent exposure but demanded prophylaxis. All were residents of rural areas.

Five raccoons exposed only members or close friends of a single household. Three raccoons exposed others in the community. In the first such incident, a baby raccoon was passed between two families and handled by many neighborhood children, exposing 16 persons. In the second incident, a rabid raccoon displayed in a store exposed 13 customers and staff. In the third incident, a science teacher encouraged a student to bring his pet raccoon to school; two students in the class required prophylaxis after the raccoon was found to be rabid.

TABLE 1—Summary Data for Eight Incidents of Actual and Potential Human Rabies Exposures from Pet Wild Raccoons, South Carolina (SC) and West Virginia (WV), 1987–1988

	Number of Persons Exposed		Type of Exposure			
State	Child[a]	Adult	Bite	Scratch or Contact with Saliva	Duration of Captivity (months)	Minimum Incubation Period[b] (months)
SC	5	8	7	6	1	1
SC	0	2	1	1	.25	.25
SC	1	0	1	0	?[c]	?[c]
SC	2	2	4	0	1	1
SC	1	1	2	0	12	1.25
WV	7	9	1	14	1	1
WV	0	1	1	0	8	3
WV	3	3	1	4	0.5	0.5
Total	19	26	18	25	3.4 (mean)	1.1 (mean)

[a] ≤15 years of age.

[b] Time between last possible contact with a potentially rabid animal and time of the raccoon's diagnosis of rabies (either onset of signs or date of laboratory testing).

[c] Unknown.

The 45 persons in the 8 incidents received 420 cc of RIG and 225 doses of HDCV at a cost of $21 092. Physician, emergency department, and clinic fees were $2622, for total direct health care costs of $23 714 or $527 per treated person.

Discussion

Keeping wild animals as pets poses a substantial risk of human rabies exposure. No vaccine can prevent rabies infection in such animals, because current rabies vaccines are not licensed for use in wild animals. As in all the incidents described in this report, owners of pet rabid animals are often not aware of their animal's infection because, at the time of capture, the animal may appear completely healthy. In addition, rabies infection in raccoons and other wild animals may have a longer subclinical course than in domestic animals.[6-8] In our series, five of eight animals had incubation periods lasting 1 month or longer. If an animal is infectious for a long period of time, both the number of persons contacting the animal and the likelihood of rabies exposure increase.

On average, a rabid pet wild animal exposes more persons to rabies than a rabid domestic animal or free-living wild animal.[9] This is probably due to longer human contact with pet wild animals than with wild animals in nature, and/or the greater attraction posed by an unusual pet such as a raccoon than is posed by more common domestic animals. In addition, pet wild animals may bite or scratch more than domestic animals, and such behavior may be more readily considered normal, resulting in delayed testing for rabies. As an extreme example of the large numbers of persons who can be exposed to a single animal, one pet rabid raccoon in Florida accounted for the prophylaxis of 172 persons at a cost of more than $64 000.[4] Moreover, many rabies exposures from wild animals may go unrecognized and untreated in children. Most dog bite injuries in this age group are not reported to health professionals[10]; the same may be true of bite injuries from wild animals.

Current legislation or regulation may be insufficient. For example, South Carolina regulations do not forbid keeping wild animals as pets. While West Virginia law allows wild animal adoption only during the hunting season for that particular species and requires a special permit, the owners of the three raccoons in West Virginia reported ignorance of these regulations. Public education is needed regarding the dangers of keeping wild animals as pets and the existence of applicable laws and regulations. Programs should target groups at highest risk, including children and residents of rural areas. ☐

References

1. Centers for Disease Control. Rabies surveillance, United States, 1988. *MMWR.* 1989;38(No. SS-1):8–9. CDC Surveillance Summaries, August 1989.
2. Deisch SL. Reported human injuries or health threats attributed to wild or exotic animals kept as pets (1971–1981). *J Am Vet Med Assoc.* 1982;180:382–383.
3. South Carolina Department of Health and Environmental Control. The ten thousand dollar raccoon. *Prev Med. Q.* 1960;3:5–7.
4. Florida Department of Health and Rehabilitative Services. Wild animals as pets: a continuing problem. *Epi-Gram.* 1987; 8:1–2.
5. Centers for Disease Control. Compendium of animal rabies control, 1990. *MMWR.* 1990;39(No. RR-4):6.
6. Jenkins SR, Winkler WG. Descriptive epidemiology from an epizootic of raccoon rabies in the middle Atlantic states, 1982–1983. *Am J Epidemiol.* 1987;126:429–437.
7. Bigler WJ, McClean RG, Trevino HA. Epizootiologic aspects of raccoon rabies in Florida. *Am J Epidemiol.* 1973;98:326–335.
8. Hubbard DR. A descriptive epidemiological study of raccoon rabies in a rural environment. *J Wildl Dis.* 1985;21:105–110.
9. Helmick CG. The epidemiology of human rabies postexposure prophylaxis, 1980–1981. *JAMA.* 1983;250:1990–1996.
10. Beck AM, Jones BA. Unreported dog bites in children. *Pubic Health Rep.* 1985;100: 315–321.

Routine Nutrition Screening Could Prevent Serious Health Problems in the Nation's Elderly, Report Says

A significant percentage of older Americans are at risk of malnutrition which could be detected and prevented through routine, inexpensive nutrition screening and greater attention to nutritional needs, according to a research report released recently.

"This excellent report lends new weight to the proposition that routine nutrition screening and care can help prevent serious illness and keep older Americans healthy and independent," said Representative Edward Roybal (D-Ca), Chair of the House Select Committee on Aging, who joined in releasing the report. "That's not only good for individuals, it's good news for a nation struggling to hold down health care costs."

The report, *Screening Older American's Nutritional Health: Current Practices and Future Possibilities,* was written by Johanna T. Dwyer, DSc, RD, Director of the USDA Frances Stern Nutrition Center at Tufts University in Boston. It is one of the most comprehensive reviews ever undertaken to examine the prevalence of malnutrition and nutrition-related problems among this nation's elderly.

"Using widely accepted criteria, a substantial proportion of older Americans have dietary intakes or diseases which place them at high risk of malnutrition," Dwyer said. "Routine nutrition screening, not only to detect the presence of malnutrition but to identify those at risk of poor nutrition, is the essential first step to improving people's nutritional status and keeping older Americans free living and in good health."

In a Foreword to the Report, Joyce T. Berry, PhD, US Commissioner on Aging, says, "Nutrition is one of many important factors affecting the health and longevity of older persons. For many low-income and minority older Americans it might be the most important."

Representative Ralph Regula (R-OH), a senior minority member of the House Select Committee on Aging, applauded Dwyer's recommendation that "screening for risk factors of malnutrition in older Americans does not need to be—nor should it be—limited to health professionals.

"Dr. Dwyer makes the case convincingly that attention to nutritional status needs to become as routine as taking our temperature or pulse," Regula said.

"The fact that nonmedical professionals and individuals themselves can employ screening techniques represents tremendous potential to keep people healthy without resorting to costly medical treatments," Regula said. "And the cost for a few minutes spent screening in a physician's office as part of a routine check-up could save many times that much in acute or chronic care treatment later."

Research has shown that people suffering from poor nutritional status have longer and more expensive hospital stays, and suffer more major complications when they are ill or injured.

Dwyer's research was commissioned by the Nutrition Screening Initiative, a five-year project focused on nutrition screening and better nutritional care among the nation's older adults.

Canine Seroprevalence and the Distribution of *Ixodes dammini* in an Area of Emerging Lyme Disease

Peter W. Rand, MD, Robert P. Smith, Jr, MD, MPH, and Eleanor H. Lacombe, BS, MT(ASCP)

ABSTRACT

This study evaluates the relative usefulness of canine serosurveys to predict risk of exposure in an area of emerging Lyme disease by comparing the distribution of canine seroprevalence with that of vector ticks. From 16 veterinary clinics throughout the State of Maine, 828 canine sera were obtained during the heartworm-testing months of April and May 1989 and measured for anti-*Borrelia* antibodies by enzyme-linked immunosorbent assay. In the same year, 1605 ticks, including 585 *Ixodes dammini*, were collected from pets, humans, small mammals, and deer. Thirty-six dogs were seropositive, 28 of which had not traveled to endemic areas. Eighty-nine percent of all seropositive dogs were from towns within 20 miles (32 km) of the coast; the great majority lived within 5 miles (8 km) of tidewater (odds ratio = 4.45, $P = .002$). Positivity varied from 17% in a southern coastal clinic to 0% in four northern clinics. Of 585 *I. dammini* identified, all but 5 (99.1%) were also from towns within 20 miles of the coast. Comparison of *I. dammini* submissions with those of another commonly found tick, *Ixodes cookei*, corroborated this predominantly coastal distribution. Canine seropositivity generally coincided with this coastal range. These data predicted areas of risk for human Lyme disease, although the prevalence of reported cases remained low. (*Am J Public Health.* 1991;81:1331–1334)

Introduction

Predicting potential areas of Lyme disease risk poses a difficult challenge for public health personnel, particularly in emerging areas with low case prevalence. Passive surveillance for reported human cases is likely to be both insensitive and of questionable specificity.[1–4] Tick vector distribution surveys based on small mammal trapping, flagging, or examination of deer are labor intensive and do not provide relative estimates of the risk of Lyme disease transmission. On the other hand, given that dogs are at greater risk of exposure than humans,[5] canine anti-*Borrelia* serosurveys offer a promising means for targeting areas for potential human risk.

The tick vector of Lyme disease, *Ixodes dammini*, was first reported in Maine in 1985.[6] Despite its subsequent establishment in coastal areas and the proximity of a hyperendemic community to the south,[7] only six cases of locally acquired Lyme disease had been reported through 1988 (unpublished data, Maine Bureau of Health Epidemiology Program, February 1991). In that year we initiated tick distribution surveys and a canine serosurvey as methods of projecting local risk of infection that might be more sensitive, efficient, timely, and economical than those involving human surveillance.

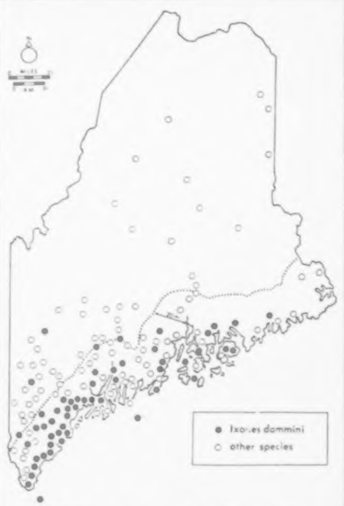

FIGURE 2—Towns of origin of identified ticks.

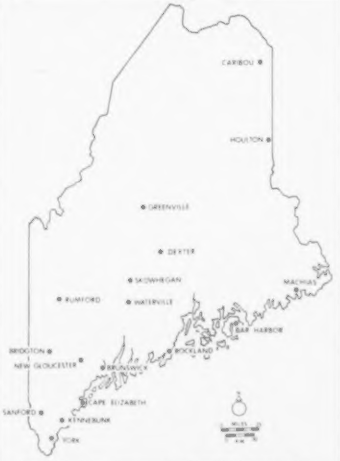

FIGURE 1—Towns of veterinary clinics cooperating in study (Dexter clinic also serves Greenville).

Materials and Methods

Ticks were submitted by veterinarians, physicians, and the public in response to published solicitations. They were also collected from natural hosts (live-trapped mice and tagged deer) by field workers and trained biologists. Tick identification was based on criteria established through

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The research for this paper was conducted at the Research Department, Maine Medical Center, Portland, Me.

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This paper was submitted to the journal January 22, 1991, and accepted with revisions May 7, 1991.

TABLE 1—Previous Canine Anti-*Borrelia* Serosurveys

Year of Study	Location	No. of Dogs	Method	% Positive	Reference
			Surveys of random asymptomatic dogs		
1984	Wisconsin	380	IFA ≥1:64	53	14
1984–1985	New Jersey	221	IFA ≥1:64	27.6 (nonendemic areas)	15
		202		42.6 (endemic areas)	
			Surveys including symptomatic dogs		
1979–1983	Southern Connecticut	210	IFA ≥1:64	28.6 (endemic areas)	16
1983, 1985	North Carolina	1002	IFA ≥1:64	3.6 (1983) 2.7 (1985)	17
1984–1985	Connecticut, New York	271	IFA ≥1:64 ELISA OD ≥0.12	66.5 (CT) 76.3 (NY)	18
1986	Northern California	345	IFA ≥1:64	26	19
1988	Texas	2400	IFA ≥1:128	5.5	20

Note. IFA = immunofluorescent assay; ELISA = enzyme-linked immunosorbent assay.

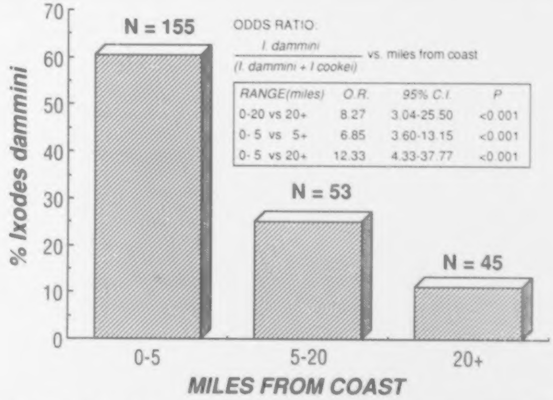


FIGURE 3—Percent *I. dammini* among *I. dammini/cookei* submissions from humans, cats, and dogs vs. miles from the coast.

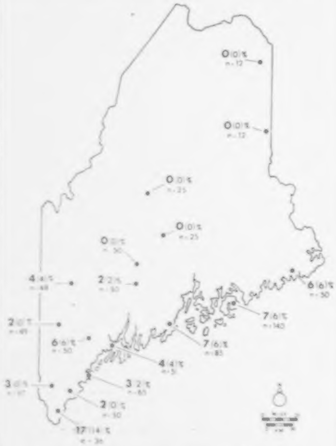


FIGURE 4—Clinic seroprevalence. Bold number is percent of all dogs in each clinic that were seropositive. Number in parentheses is percent of untraveled dogs that were seropositive. n = total number of dogs studied in each clinic.

standard keys,[8–12] verified as necessary by the Smithsonian Institution, Washington, DC.

Sixteen high-volume veterinary clinics throughout Maine (Figure 1) were invited to collaborate on the basis of their proximity to recognized endemic regions (three southernmost clinics), to areas — primarily coastal — where specimens of *I. dammini* had been collected (five clinics from Cape Elizabeth to Machias), and to inland and northern regions with little or no history of the disease or its vector (eight clinics, one serving two towns). Each clinic was provided with sampling materials and questionnaires for each pet owner. Veterinarians were asked to provide an additional 1 ml of serum from 50 or more dogs tested for heartworm microfilaria between April 1 and June 1, 1989. The only selection criteria imposed were that dogs be over 6 months old, lifetime residents of Maine, and allowed, at least sometimes, to run in fields or woods. Frozen sera were shipped to Tufts University Veterinary Diagnostic Laboratory for analysis of antibodies to *B. burgdorferi* by enzyme-linked immunosorbent assay.[13] Questionnaire items included the dog's breed, age, sex, principal function, degree of confinement, and history of travel to endemic areas. Owners were also asked if their pets had previously been lame or treated for Lyme disease.

Results

Tick Collection

Of 1605 ticks examined in 1989, 585 were *I. dammini*. Of these, 218 were submitted and 182 were collected from 789 live-trapped small mammals, 78 from mist-netted birds, 39 from flagging, and 68 from 30 of 227 examined deer. Towns from which *I. dammini* and other tick species were received are shown in Figure 2. There is notable clustering along the coast: all but 5 of 53 towns producing *I. dammini* are within 20 miles of the tidal water.

To demonstrate that the high numbers of *I. dammini* submitted from coastal regions by the public were not the result of increased population density or vigilance in these areas, submissions of this tick

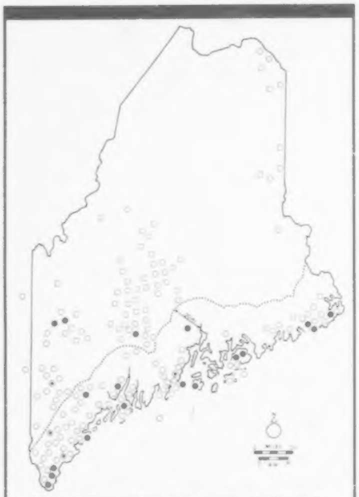
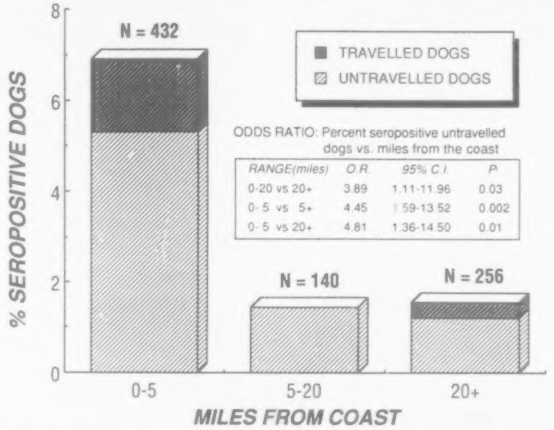


FIGURE 5—Home towns of dogs in the study. Open circles (○): no dogs positive; solid circles (●): positive untraveled dogs present; dotted circles (⊙): positive traveled dog present.



ODDS RATIO: Percent seropositive untravelled dogs vs. miles from the coast

RANGE(miles)	O.R.	95% C.I.	P.
0-20 vs 20+	3.89	1.11-11.96	0.03
0-5 vs 5+	4.45	1.59-13.52	0.002
0-5 vs 20+	4.81	1.36-14.50	0.01

FIGURE 6—Percent of traveled and untraveled dogs positive for anti-*Borrelia* antibodies vs. miles from the coast.

were compared to submissions of *Ixodes cookei,* which closely resembles *I. dammini,* is widely distributed in Maine, and is found more frequently on humans than *I. dammini.* Figure 3 shows that *I. dammini* comprised 64% of the combined *I. dammini/cookei* submissions within 5 miles of the coast, but only 10% of the submissions beyond 20 miles.

Canine Serosurvey

Sera from 828 dogs were received from 149 towns representing all counties. Thirty-six samples were positive, 28 of which were from untraveled dogs. Clinics with seropositive dogs were limited to primarily coastal areas in the southern half of the state (Figure 4). Highest seroprevalence (17%) occurred in dogs from the southernmost clinic. Positive untraveled dogs were also found in two inland communities, both on rivers but in nonendemic counties. No positivity was found among 102 dogs in the northern half of the state.

Figure 5 demonstrates that, with the exception of four towns, each of which had one positive dog, the home towns of positive dogs were all within a line arbitrarily drawn 20 miles (32.2 km) from tidal saltwater. Thirty-two of all 36 positive dogs (89%) and 25 of 28 traveled dogs (89%) were within this coastal zone; in fact, the great majority of both untraveled and traveled positive dogs were within the first 5 miles (8 km) of the coast, as shown in Figure 6.

Among the positive dogs there were more retrievers than in the negative group (55% vs 40%), median age was higher (6.3 vs 4.7 years), and a greater percentage of dogs were allowed to run free (61% vs 54%). The percentage of asymptomatic animals in each group was not significantly different.

Discussion

Comparison with previous studies (Table 1) indicates that even the highest level of canine seropositivity in Maine is less than that in other endemic states. While this may reflect relatively lower tick densities and infection rates, the presence of canine seropositivity so geographically coincident with the distribution of *I. dammini* cannot be overlooked as an indication of potential risk to humans in Maine or in other emergent areas. Subsequent to this study, eight additional cases of locally acquired human Lyme disease have been confirmed in Maine, all from coastal counties. Thus, both canine serology and vector tick distribution surveys have identified areas of human risk in advance of substantiation of that risk through passive human surveillance.

Canine anti-*Borrelia* serosurveys are simple, efficient, and economical. Coordination of sampling with annual heartworm testing is particularly advantageous because it provides a large, readily available population of dogs with known histories and a standard sampling time (April and May)—an important consideration given the marked seasonal variations in canine seropositivity that have been observed.[14, 18] The introduction of a canine Lyme disease vaccine in June 1990, however, may impair the future usefulness of dog serosurveys since dogs who receive it must be excluded from serosurveys. Anything beyond minimal vaccine use in an emergent area might introduce enough selection bias to limit the epidemiologic relevance of canine serosurvey data. ☐

Acknowledgments

This work was supported by grants from the Elmina B. Sewall Foundation, Davis Family Foundation, Simmons Foundation, and L. L. Bean, Inc. The authors gratefully acknowledge the assistance of Drs. Merle Weber and Joanne Lindenmayer of Tufts Veterinary Diagnostic Laboratory where the antibody testing was performed. They also thank State Veterinarian Dr. Chip Ridkey for his organizational advice, the veterinarians and staff of the 16 clinics for making this study possible through their interest and support, and Dr. W. Douglas Thompson for statistical analysis. Dr. Richard Dearborn of the Maine Insect and Disease Laboratory contributed data on tick distribution, and Dr. James E. Keirans, Research Entomologist at the Smithsonian Institution, confirmed tick species identification. Dr. Kathleen Gensheimer, epidemiologist, Maine Bureau of Health, provided data on reported human cases of Lyme disease.

References

1. Thacker SB, Choi K, Brachman P. The surveillance of infectious diseases. *JAMA.* 1983;249:1081–1085.
2. Chorba TL, Berkelman RL, Safford SK, Gibbs NP, Hull HF. Mandatory reporting of infectious diseases by clinicians. *JAMA.* 1989;262:3018–3025.

3. Magnarelli LA. Serological diagnosis of Lyme disease. *Ann NY Acad Sci.* 1988;539:154–161.
4. Hedberg CW, Osterholm MT, MacDonald KL, White KE. An interlaboratory study of antibody to *Borrelia burgdorferi. J Infect Dis.* 1987;155:1325–1327.
5. Eng TR, Wilson ML, Spielman A, Lastavica CC. Greater risk of *Borrelia burgdorferi* infection in dogs than in people. *J Infect Dis.* 1988;158:1410–1411.
6. Anderson JF, Magnarelli LA, McAninch JB. *Ixodes dammini* and *Borelia burgdorferi* in northern New England and upstate New York. *J Parasitol.* 1987;73:419–421.
7. Lastavica C, Wilson ML, Berardi VP, Spielman A, Deblinger R. Rapid emergence of focal epidemic of Lyme disease in coastal Massachusetts. *N Engl J Med.* 1989; 320:133–37.
8. Keirans JE, Clifford CM. The genus *Ixodes* in the U.S.: a scanning electron microscope study and key to adults. *J Med Entomol.* 1978;Suppl 2:1–149.
9. Clifford CM, Anastos G, Elbi A. The larval *Ixodes* ticks of the eastern United States (Acarina-Ixodidae). *Entomol Soc Am.* 1965;2:213–237.
10. Keirans JE, Litwak TR. Pictorial key to the adults of hard ticks, family Ixodidae (Ixodida: Ixodoidea), east of the Mississippi River. *J Med Entomol.* 1989;26:435–448.
11. Cooley RA, Kohls GM. The genus *Ixodes* in North America. *Natl Inst Health Bull.* 1945;184:1–246.
12. Spielman A, Clifford VM, Piesman J, Corwin MD. Human babesiosis on Nantucket Island, U.S.A.: description of a vector, *Ixodes* (Ixodes) *dammini,* N. Sp. (Acarina: Ixodidae). *J Med Entomol.* 1979;15:218–234.
13. Lindenmayer J, Weber M, Bryant J, Marquez E, Onderdonk A. Comparison of indirect immunofluorescent-antibody assay, enzyme-linked immunosorbent assay, and Western immunoblot for the diagnosis of Lyme disease in dogs. *J Clin Microbiol.* 1990;28:92–96.
14. Burgess EC. Natural exposure of Wisconsin dogs to the Lyme disease spirochete. *Lab Anim Sci.* 1986;36:288–290.
15. Schulze TL, Bosler EM, Shisler JK, Ware IC, Lakat MF, Parkin WE. Prevalence of canine Lyme disease from an endemic area as determined by serosurvey. *Zbl Bakt Hyg.* 1986;263:427–434.
16. Magnarelli LA, Anderson JF, Kaufmann AF, Lieberman LL, Whitney GD. Borreliosis in dogs from southern Connecticut. *J Am Vet Med Assoc.* 1985;186:955–959.
17. Greene RT, Levine JF, Breitschwerdt EB, Berkhoff, HA. Antibodies to *Borrelia burgdorferi* in dogs in North Carolina. *Am J Vet Res.* 1988;49:473–476.
18. Magnarelli LA, Anderson JF, Schreier AB, Ficke CM. Clinical and serologic studies of canine borreliosis. *J Am Vet Med Assoc.* 1987;191:1089–1094.
19. Teitler J, Madigan J, DeRock E, Pedersen N, Carpenter T, Franti C. Prevalence of *Borrelia burgdorferi* antibodies in dogs in Northern California. *Ann NY Acad Sci.* 1988;539:500–503.
20. Cohen ND, Carter CN, Thomas MA Jr., Angulo AB, Eugster, AK. Clinical and epizootiological characteristics of dogs seropositive to *Borrelia burgdorferi* in Texas: 110 cases (1988). *J Am Vet Med Assoc.* 1990;197:893–898.

NCHS to Publish New Data on Persons With and Without Health Care Coverage

The new study is entitled "Characteristics of Persons With and Without Health Care Coverage: United States, 1989" (*Advance Data* No. 201, National Center for Health Statistics, Centers for Disease Control, PHS, DHHS).

Highlights

An estimated 33.9 million Americans were without health care coverage in 1989. Health care coverage includes private health insurance, Medicare, public assistance (primarily Medicaid), and military or Veteran's Administration health benefits. Data were collected in the 1989 National Health Interview Survey, a nationwide household interview survey of the Nation's civilian, noninstitutionalized population.

People who lacked health care coverage included almost 40% of unemployed workers 18 years old or older (38.3 percent) and almost a third of people living below the poverty level (32.5 percent).

About 76.1% of the population were covered by private health insurance; 12.6% through Medicare; 6.2% by public assistance health care programs; and 2.6% were covered by military and/or Veterans' Administration health care programs.

Those without health care coverage increased from 13% in 1984 to 13.9% of the population in 1989. Although the proportion of people without coverage is greater for those below the poverty level, the increase in lack of health care coverage was greater for those above the poverty level during this period.

Situational Factors Associated with AIDS Risk Behavior Lapses and Coping Strategies Used by Gay Men Who Successfully Avoid Lapses

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ABSTRACT

While most gay men have reduced behavior practices at high risk for HIV infection, there is growing evidence that many also lapse to unsafe sex. This study examined situational factors related to risk behavior lapses as well as coping strategies used by men who successfully resist lapse urges.

A convenience sample of 470 men patronizing gay bars or attending social organization meetings in four cities was surveyed. Forty-five percent of men were classified as "lapsers" (those who had had unprotected anal intercourse in the previous 6 months) and 24% were classified as "resisters" (those who successfully resisted urges to engage in this behavior). All provided information concerning the importance of factors related to the most recent occurrence of either unsafe sex or resisting unsafe urges.

Most episodes of unsafe sex occurred outside monogamous relationships and with partners of unknown HIV serostatus, although simply inquiring about partner serostatus was relatively common. Lapsers rated affectionate feelings and wishing to please a partner as well as spontaneity of unsafe sex as the most important situational factors surrounding high-risk behavior. Resisters of unsafe sex urges reported active cognitive self-guidance, experience in safe sex, and recall of both AIDS fears and safety benefits as their most important coping strategies.

Gay men who continue high-risk behavior may be overrelying on partner reports of negative serostatus. Lapse prevention approaches tailored to situations that create increased risk vulnerability must be developed. Teaching skills already used by men who successfully resist unsafe sex urges might be one approach. (*Am J Public Health.* 1991;81:1335–1338)

Introduction

Gay men in AIDS epicenters have made substantial risk reduction behavior changes.[1-5] However, these changes are difficult for many individuals to sustain. One third of homosexual men in Chicago who successfully initiate risk reduction later resume high-risk practices[6,7] Cross-sectional studies indicate that 30% to 40% of gay men in other cities still report engaging in unprotected anal intercourse in a given 3-month period.[8-10] Relapse is a concern even among gay men enrolled in San Francisco AIDS behavioral research cohorts, presumably one of the most AIDS-sensitized samples in the United States.[11]

Homosexual men who continue to engage in high-risk sex even occasionally are in great danger of contracting HIV infection. Gay men in large cities who have unprotected receptive anal intercourse with a single partner over 1 year are three times more likely to seroconvert than men who refrain from this activity and, with five partners, are 18 times more likely to develop HIV infection.[12] While lapses are the rule rather than the exception following initiation of change in all health behavior areas,[13-15] we were aware of no other population or health area where there is such a small margin for error and where even infrequent lapses carry such potentially grave health consequences.

Risk behavior relapse among gay men is predicted by younger age, lower education level, excessive intoxicant use, frequency of past risk behavior with multiple partners, and high reported past enjoyment associated with risky practices.[8,11,16,17] The present study more closely evaluated specific situational and social context factors surrounding risk behavior lapses as well as the coping strategies used by gay men who report successfully resisting high-risk behavior urges.

Method

Subjects

Subjects were 406 men who patronized gay bars and 64 men who attended social meetings of gay organizations in the spring of 1990 in four cities: Memphis, Tenn, Tampa, Fla, Mobile, Ala, and Binghamton, NY. Approximately 75% of men who were approached agreed to answer questions anonymously. The mean age of respondents was 31.0 years (SD=8.6) and the mean education level was 14.9 years (SD=2.2); 91% of the sample was White and 9% was Black, Hispanic, or of other racial or ethnic backgrounds. The city samples did not differ significantly in demographic characteristics.

Measures

Respondents described their sexual behavior over the preceding 6 months including frequency and number of partners for unprotected anal intercourse, both receptive and insertive. Forty-two percent of men who engaged in unprotected anal intercourse were the insertive partner, 35% the receptive partner, and 23% were both insertive and receptive during their most recent behavior. Multivariate analysis of variance revealed no significant difference between those who were the in-

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This paper was submitted to the journal August 13, 1990, and accepted with revisions March 7, 1991.

TABLE 1—Situations Associated with the Most Recent Occurrence of High-Risk Behavior among Gay Men Who Report Unsafe Sex during the Previous 6 Months

Situational Descriptor	Mean (SD) Importance Rating
This partner was someone special to me	2.7 (1.3)
I wanted to please this partner	2.2 (1.3)
Once sex began, we got caught up in the passion of the moment	2.2 (1.2)
No condoms were nearby at the time of sex	2.0 (1.3)
Using a condom seemed impersonal	1.9 (1.1)
Using a condom would have reduced sensation/pleasure	1.9 (1.2)
Stopping to use a condom would have interrupted sex	1.8 (1.1)
I had a strong urge to have anal intercourse without a condom	1.8 (1.1)
It was hard for me to refuse having anal intercourse	1.8 (1.1)
It was a long time since I had anal intercourse without a condom	1.8 (1.1)
I had a lot to drink or used drugs and wasn't clearheaded	1.7 (1.1)
Condom use implies a lack of trust	1.5 (0.9)
It was embarrassing for me to suggest using a condom	1.5 (0.9)
I felt no control over what we did	1.5 (1.0)
I am not able to change my sex practices	1.5 (0.9)
I was feeling depressed, lonely, or upset at the time	1.4 (0.9)
This partner pressured me not to use a condom	1.4 (0.9)
It was too hard to get the condom package open	1.3 (0.7)
I didn't have the energy to use a condom	1.3 (0.6)
I pressured this partner not to use a condom	1.2 (0.6)
I was too embarrassed to buy condoms	1.2 (0.7)

Note. Importance ratings made on scale from 1=not a factor to 4=a big factor.

TABLE 2—Coping Strategies Reported to Be Used by Gay Men Who Successfully Resisted Urges to Engage in Unsafe Sex during the Previous 6 Months

Coping Strategy	Mean (SD) Importance Rating
I remembered information about AIDS and safer sex	3.6 (0.9)
I thought about how important it is to be in good health	3.5 (0.9)
I was afraid of getting (or giving) the AIDS virus	3.5 (1.0)
I reminded myself to stay safe before or during sex	3.4 (1.0)
I knew how good I'd feel about myself later if I stayed safe	3.3 (1.1)
I didn't want to worry about AIDS after sex	3.3 (1.1)
I am experienced in safer sex	3.3 (1.0)
I decided ahead of time what I would or would not be willing to do	3.2 (1.0)
I actively guided our actions to stay safe	3.1 (1.0)
The scare of knowing people with AIDS motivated me to be safe	3.1 (1.2)
I told this partner we needed to practice safer sex	2.9 (1.1)
I wanted this partner to know I was health conscious	2.9 (1.2)
I kept condoms nearby	2.9 (1.2)
I told this partner I didn't want to have unprotected intercourse	2.9 (1.2)
We switched from something that was getting risky to something safer	2.5 (1.2)
Safer sex was as good as intercourse without a condom	2.5 (1.2)
Safer sex was expected by this partner	2.4 (1.3)
I didn't drink or use drugs much before sex so I could be clearheaded	2.3 (1.1)
This partner told me he didn't want to have unprotected intercourse	2.3 (1.2)
This partner told me that we needed to practice safer sex	2.2 (1.3)
I didn't want other people to find out that I might have unsafe sex	2.0 (1.2)

Note. Importance ratings made on scale from 1=not a factor to 4=a big factor.

sertive vs receptive partner in importance ratings for lapse situation items, so the insertive and receptive groups were combined for all data analyses. They were also questioned about number of partners and frequency for anal intercourse using condoms and about safer sex practices without penetration such as mutual masturbation and frottage (body rubbing). In addition, all men reported whether they presently had condoms in their pocket, car, and home.

Respondents who reported engaging in unprotected anal intercourse in the previous 6 months ("lapsers," n=209, 45% of the sample) described, for the most recent occurrence of high-risk behavior, exclusivity of relationship with the partner, knowledge of own and partner's HIV serostatus, difficulty in maintaining safer sex practices, and percentage of anal intercourse occasions when condoms were used in the previous year. These men then rated the importance of 21 situational factors associated with the most recent occasion of high-risk behavior. As Table 1 shows, these items assessed environmental and setting factors, feelings toward the partner, coercion, physiological and emotional state, management of sudden urges, and self-control.

A second set of men were those who reported successfully resisting temptation to engage in unprotected anal intercourse in the previous 6 months ("resisters," n=116, 24% of the sample). (Men who reported no occurrence of unprotected anal intercourse *and* no temptation to engage in the practice were not studied here.) This group of men rated the importance of 21 coping strategies that may have been used on the most recent occasion when high-risk urges were resisted. Table 2 lists these items, which include risk reduction self-prompts; environmental modifications; active use of alternative behaviors; cognitive statements emphasizing benefits, fears, or beliefs of personal efficacy; and social expectations to avoid risk.

Results

As Table 3 shows, men reporting high-risk behavior were younger than those who reported resisting unsafe sex urges, had less education, and had had more male sexual partners in the previous 6 months. Groups were comparable in reporting having condoms in their pocket, car, or home and reported similar difficulty levels for remaining safe during sex.

Table 1 presents the mean importance rating for each situational descriptor associated with high-risk behavior by men who reported lapses, ranked from most to least important. Factors most critical to lapses were positive emotional feelings for the partner, wishing to please the partner, not having condoms nearby at the time of sex, becoming "caught up in passion of the moment," and negative connotations about condom use. As Table 2 shows, men who resisted high-risk urges reported the following as their most important coping strategies: cognitive reminders to re-

TABLE 3—Characteristics of Men Who Lapsed to Unsafe Sex and Men Who Successfully Resisted Relapse Urges during the Previous 6 Months

Characteristic	Lapsers (n=209)	Resisters (n=116)	95% CI[a]
Mean age in years (SD)	29.4 (7.6)	31.9 (8.4)	29.5–31.3
Mean educational level in years (SD)	14.6 (2.1)	15.6 (2.0)	14.7–15.2
Mean number of different male sexual partners in previous 6 months (SD)	4.5 (7.6)	3.4 (4.2)	3.4–4.8
Percentage of men reporting having condoms in their			
Pocket or wallet	29	27	—
Car	42	37	—
Home	84	93	—
Mean personal difficulty rating for remaining safe during sex (SD)[b]	2.6 (1.2)	2.5 (1.1)	2.3–2.8
Mean percentage of intercourse occasions when condoms were used during previous year	44	—	—

[a] CI = confidence interval.

[b] Difficulty ratings made on a 5-point Likert scale from 1=very easy to stay safe to 5=very difficult to stay safe during sex.

TABLE 4—Relationship between Knowledge of Own HIV Serostatus and Knowledge of Sexual Partner's Serostatus among Men Reporting Occurrence of Unprotected Anal Intercourse

	Knowledge of Partner's HIV Serostatus			
Knowledge of Own HIV Serostatus	Didn't Know Partner Serostatus (n=97)	Partner Knew Was HIV Positive (n=5)	Partner Said He Was HIV Negative (n=61)	Saw Evidence Partner Was HIV Negative (n=35)
Not tested (n=55)	44 (22%)	1 (0.5%)	9 (4.5%)	1 (0.5%)
HIV positive (n=10)	7 (4%)	1 (0.5%)	2 (1%)	0 (0%)
HIV negative (n=133)	46 (23%)	3 (1.5%)	50 (25%)	34 (17%)

Note. Chi-square=40.1, df=6, $P < .0001$ for the association of knowledge of own serostatus and knowledge of partner's serostatus.

main safe during sex, self-statements regarding fear and avoiding worry, identifying the benefits of safety and good health, and experience and competence in actively guiding safer sex.

To examine the context of high-risk episodes, relationship status and serostatus knowledge between partners were also explored. Most high-risk activity (57%) took place between nonexclusive and casual partners; 43% occurred with partners described as exclusive, although the mean duration of the relationships was only 12.5 months. Table 4 shows the distribution for high-risk behavior in relation to knowledge of one's own and one's partner's HIV serostatus. Nearly half of the respondents who engaged in high-risk behavior had no knowledge of their partner's HIV serostatus; an additional 31% reported that the partner simply said he was HIV negative. In less than 18% of instances did respondents report first-hand knowledge of partner's HIV-negative status and, even when high-risk behavior occurred with a partner described as exclusive, two thirds of these men said they did not know both their own and their partner's serostatus. In general, knowledge of one's own negative status was related to a greater likelihood of inquiring about a partner's HIV status.

Discussion

Nearly one half of respondents in this sample had engaged in unprotected anal intercourse in the previous 6 months, usually with nonexclusive partners of unknown HIV serostatus. Even men who engaged in high-risk behavior appeared cognizant of risk: well over 80% said they had condoms at home and lapsers used condoms almost half the time. In addition to the association of risk behavior with affectionate feelings toward the partner and spontaneity of risky sexual practices, a large proportion of lapsers accepted partner claims of negative HIV status or their own past negative result. Among persons who continue to engage in high-risk behavior, past negative HIV results of self or partner, even if accurate, carry little current protective value. Approaches are needed that better prepare gay men to anticipate and handle lapse temptations, counter misconceptions that may perpetuate vulnerability to lapses, and stress the importance of maintaining consistent behavior change. These results indicate that resistance to lapses is related to active use of cognitive self-guidance, recall of both AIDS fears and safety benefits, and experience in safer sex. Although the current study is limited by its use of a convenience sample of unknown generalizability, respondent self-reports of behavior, and analysis only of "within-group" patterns, it highlights the need for further study of ways to prevent risk lapses in populations vulnerable to HIV infection. ☐

Acknowledgments

This research was supported by National Institute of Mental Health grant R01-MH42908. The authors thank the following persons for data collection assistance: Rick Bray, Chris Miller, AID to End AIDS Committee—Memphis, Rev. Lew Broyles, Bob Fancher, Tommy Moore, and Risky Business—Binghamton, NY.

References

1. Martin JL. Impact of AIDS on gay male sexual behavior patterns in New York City. *Am J Public Health*. 1987;77:578–581.
2. Becker MA, Joseph JG. AIDS and behavior change to reduce risk: a review. *Am J Public Health*. 1988;78:394–410.
3. Martin JL, Garcia MA, Beatrice ST. Sexual behavior changes and HIV antibody in a cohort of New York City gay men. *Am J Public Health*. 1989;79:501–503.
4. McKusick L, Horstman W, Coates TJ. AIDS and sexual behavior reported by gay men in San Francisco. *Am J Public Health*. 1985;75:493–496.
5. McKusick L, Wiley JA, Coates TJ, et al. Reported changes in the sexual behavior of men at risk for AIDS: San Francisco 1982–1984: the AIDS Behavior Research Project. *Public Health Rep*. 1985;100:622–629.
6. Joseph JG, Montgomery SG, Emmons C,

et al. Perceived risk of AIDS: assessing the behavioral and psychosocial consequences in a cohort of gay men. *J App Soc Psychol*. 1987;17:231–250.

7. Joseph JG, Montgomery SG, Kessler RC, et al. Determinants of high risk behavior and recidivism in gay men. Presented at the 4th International Conference on AIDS; Stockholm, Sweden; June, 1988.
8. Kelly JA, St. Lawrence JS, Brasfield TL, et al. Psychological factors which predict AIDS high-risk versus AIDS precautionary behavior. *J Consult Clin Psychol*. 1990;58:117–120.
9. Kelly JA, St. Lawrence JS, Brasfield TL, et al. AIDS risk behavior patterns among gay men in small cities. *Am J Public Health*. 1990;80:416–418.
10. St. Lawrence JS, Hood HV, Brasfield TL, et al. Differences in gay men's AIDS risk knowledge and behavior patterns in high and low AIDS prevalence cities. *Public Health Rep*. 1989;104:391–395.
11. Ekstrand ML, Coates TJ. Maintenance of safer sexual behaviors and predictors of risky sex. *Am J Public Health*. 1990;80:973–977.
12. Kingsley LA, Detels R, Kaslow R, et al. Risk factors for seroconversion to human immunodeficiency virus among male homosexuals. *Lancet*. 1987;1:345–349.
13. Hunt WA, Matarazzo JD. Three years later: recent developments in the experimental modifications of smoking behavior. *J Abnorm Psychol*. 1973;81:107–114.
14. Marlatt GA, Gordon JR. Determinants of relapse: implications for the maintenance of behavior change. In: Davidson PO, Davidson SM, eds. *Behavioral Medicine: Changing Health Life Styles*. Elmsford, NY: Pergamon; 1980:410–452.
15. Marlatt GA, Gordon JR. *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviors*. New York, NY: Guilford Press; 1985.
16. Kelly JA, St. Lawrence JS, Brasfield TL. Predictors of vulnerability to AIDS risk behavior relapse. *J Consult Clin Psychol*. 1990;59:163–166.
17. Stall R, McKusick L, Wiley J, et al. Alcohol and drug use during sexual activity and compliance with safe sex guidelines for AIDS: the AIDS Behavioral Research Project. *Health Educ Q*. 1986;13:359–371.

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